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by

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PRESIDENT'S ADDRESS

THE GRADUATE FORTNIGHT

1847—CENTENNIAL OF THE ACADEMY—1947

BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

NOVEMBER 1947

OPENING ADDRESS*

Twentieth Graduate Fortnight

GEORGE BAEHR

President, The New York Academy of Medicine

It is now twenty years since The New York Academy of Medicine organized its first Graduate Fortnight in response to the need of physicians of the metropolitan area and of adjacent states for an annual review of the major advances in one important branch of medicine. Each year a new subject has been chosen, each year the lectures, demonstrations, hospital clinics, round table conferences, and exhibits have been better than ever before, and each year the growing attendance by several thousands of visiting physicians testifies to their appreciation of the educational value of the Fortnight. Experts from many parts of the country have generously contributed their knowledge and experience to that of our own professional and scientific leaders until the Fortnight has become one of the great intellectual treats of our country.

Throughout the year, the monthly stated meetings of the whole Academy and the monthly meetings of the twelve clinical and scientific Sections of the Academy bring to the medical profession of this area the newest information upon the important subjects of the day and

* October 6, 1947.

introduce to them many of the most important recent contributors to the advancement of the medical sciences. Other special lectures and demonstrations are given whenever opportune at other times during the year. Many clinical and scientific societies affiliated with the Academy hold their meetings within the building so that on some evenings as many as four or five meetings on different subjects may be going on simultaneously. To add still further to the educational opportunities of the medical profession, the Academy conducts, as you know, one of the largest and most used medical libraries in the world, which is freely open to all physicians and to the public until 5 in the afternoon. The Academy also maintains a well staffed educational bureau for physicians from this country, from Latin America and from abroad who seek information concerning educational opportunities in municipal and voluntary hospitals and teaching centers of this great city. For the benefit of visiting surgeons, a bulletin is published daily of the operations and the operating surgeons in every important hospital in the city so that anyone can observe any operation he may desire to witness on almost any day in the week.

These and other educational activities of the Academy are designed to make New York City the medical capital as it is already the political capital of the world. The physicians of this part of the country are the chief beneficiaries from these services, for they have the constant opportunity to enjoy them. I often wonder how many of you give a thought to the question where the money comes from for the support of all this. The library alone requires an expenditure of almost $135,000 a year. You will be surprised to know that the dues from Fellows and Associate Fellows are only sufficient to defray one-fifth of the annual operating budget of the Academy. Part of the remainder is met out of income from endowments and bequests. But a substantial part of the annual budget depends upon gifts from the public and from physicians who appreciate the work which the Academy is doing in the fields of medical education, public health, public education and through our great library.

There is, perhaps, not a physician in this city who has not benefitted from the Academy's facilities and activities, and through the medical profession the entire public has been well served for more than one hundred years. Like all institutions of learning, our operating costs have increased greatly in recent years due to essential increases in salaries,

yet income has remained stationary. More than ever before, we must look to the public and to the medical profession for assistance in carrying their share of the burden.

This Graduate Fortnight will demonstrate that the Academy has not stinted because of increasing costs nor reduced the quality and scope of its service to the profession and to the public. Experts have again been brought from other centers of education and research. The exhibits on the subjects of the Fortnight, the Diseases of Metabolism and of the Endocrine Glands, are more extensive and more carefully planned than ever before. We are deeply indebted to the Subcommittee on the Graduate Fortnight and, especially, to its chairman, Dr. Milton J. Raisbeck, to the director of scientific exhibits, Dr. Alfred Angrist, to Dr. B. S. Oppenheimer, chairman of the Committee on Hospital Clinics, and to Dr. Charles F. Tenney, chairman of the Committee on Panel Discussions, for their devoted and valuable work. We are also in debt to the coöperating hospitals which have again organized a series of public clinics for every afternoon throughout the Fortnight. Our one regret is that the absence of permanent exhibition space makes it impossible to continue the exhibits beyond the period of two weeks and that this invaluable teaching material must then be dismantled and disbursed.

With this expression of our deep appreciation for the labor of the many contributors to the forthcoming lectures, clinics, demonstrations, panel discussions, and scientific exhibits, I now officially open this Twentieth Graduate Fortnight of the Academy.

PSYCHOSOMATIC ASPECTS OF ALLERGIC DISORDERS*

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PSYCHOSOMATIC is a new term, but it describes an approach to medicine as old as the art of healing itself. It is not a new specialty but is rather a point of view which applies to all aspects of medicine and surgery. It does not mean less study of the soma; it only means more study of the psyche. Its subject matter is founded on the important advances in physical medicine as well as on the biologically oriented psychology of Freud, without whose epochal discoveries no work on psychosomatic medicine could be attempted. It is not a new discovery, but rather a reaffirmation of the ancient principle that the mind and the body are interactive and interdependent, a principle that has always guided the intelligent general practitioner. As a science, psychosomatic medicine aims at discovering the precise nature of this relationship.

Allergy and psychosomatic medicine have much in common. The allergic population and the neurotic population are so numerous that they must overlap. Therefore, if for no other reason, these disorders exist together in many people.

There are other reasons why allergists and those interested in psychosomatic medicine should be interested in one another's problems. The subjects of allergy and psychosomatic medicine coincide in many ways. Applied in an empirical way for many years, both subjects were established on a firm scientific footing after World War I and both saw a more complete integration into general medicine in World War II. Utilized to some extent by many practitioners, both subjects must become a part of the understanding of all physicians. In order to accomplish this, increased facilities must be established in medical education for undergraduates and graduates. But they have more in common than this. Both have been exploited by irregular practitioners when orthodox medicine has disdained to endorse them, and both have

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potentialities for doing harm as well as good.

In addition to these aspects which they have in common, there is an intimate relation between them. Psychosomatic medicine would seem to have a special application to allergy. It has long been recognized that emotional factors entered into the problems of allergy, but up until recently we have lacked any exact methods of measuring these effects.

Within the last decade, however, medicine has succeeded to a considerable extent in substituting scientific principles of psychopathology and psychotherapy for intuition—generally spoken of as the art of medicine—in regard to many medical problems, and this rapidly increasing body of knowledge is now referred to as psychosomatic medicine. The subject received great impetus from military medicine because of the large number of neuropsychiatric and psychosomatic problems necessitating rejection or discharge from the service. Therefore, it would seem timely to apply some of these principles to the study of problems in allergy.

The Psychosomatic Point of View

When a person gets sick he is sick all over, that is, the body and mind are one, and he gets sick for a variety of reasons, physical and psychic. In other words, it is usually not one thing that determines illness; there are multiple factors acting together. Halliday[1] points out that in our approach to illness we must think: (1) What kind of a *person* are we dealing with (inherited and acquired characteristics, physical and psychologic); (2) What has he *met* (germs, allergens, or emotionally disturbing events); (3) What has *happened* (the physiologic mechanism or pathogenesis of the disorder). For example, allergic responses occur when a prepared organism, possessing certain physical and psychologic characteristics, meets certain elements, physiologic and psychologic. In some allergic disorders a single preponderant factor may be largely responsible, as for example in pollen hay fever. In others, such as asthma, there are frequently multiple interrelated factors, allergens and psychic disturbances, which act in a complementary fashion to produce the disorder.

The psychosomatic point of view is that the psyche enters, or better emerges, as one of the several factors which, acting together, disturb the function of the organism. We will leave aside the great problem of

how much of the illness depends upon constitutional factors and to what extent special life experiences are responsible. It is the old question of heredity and environment, nature and nurture, and we have no way of delimiting them with exact measurements.

In general, physicians look upon the problem of psychic factors in illness in several ways. There is a group which considers the physical factors as being all important and indeed wholly responsible for any personality changes–"Why shouldn't he be nervous after having had such a serious disease so long." This thinking applies especially to chronic disorders such as asthma. At the opposite pole are the protagonists for psychogenesis–the psyche is all important in the determination of chronic illness. In between are those who believe that physical and psychologic aspects are but different phases of the disordered constitution, perhaps parallel manifestations of the same basic fault, existing together and related to one another. As Kubie[2] states, psychologic forces and somatic manifestations may have their roots in the same unconscious processes which discharge partly on the level of psychic representation through thoughts and feelings and partly on the physiologic level through the autonomic nervous system. This is the viewpoint presented here. We are not interested in proving psychogenesis, we are only interested in studying the psychic factor in illness, just as we study physical factors, and relate them if possible. We would like the psyche to be treated with as much respect as germs or allergens.

Just as we insist on this unity regarding the nature of illness–the body and mind are one–so do we urge that the means of investigation are two–physical and psychologic–and that their simultaneous application represents psychosomatic diagnosis.[3] And they are applied simultaneously although their application is often unconscious and unscientific. The physician in his usual approach to the patient, using the orthodox methods of medical history, physical examination, and laboratory studies, at the same time makes many intuitive observations about the unconscious mental processes of the patient as exhibited in his language and behavior. What we suggest is that order be brought into this intuitive process. We now have enough understanding so that we can begin to forge a scientific instrument in the exploration of unconscious processes. In other words we are trying to make the rules regarding psychologic exploration as exact as the rules of physical investigation.

Psychosomatic diagnosis depends much more upon the history than it does upon physical examination or laboratory studies. This is especially true in regard to psychosomatic affections.* The faulty concept of functional versus organic disease and the necessity for giving up the "either-or" diagnostic approach have been discussed elsewhere.[4] Personality study will show that psychosomatic disorders have their own distinctive features and that diagnosis must be established on positive data from psychologic study as well as negative data from an organic standpoint.

Rather than be satisfied with loose terminology—neurogenic factors, emotional upsets, physical and mental fatigue, emotional stress and strain—we must make an effort to define the specific emotional problem and relate it to the total personality makeup of the individual.

Just as we try to establish certain postulates for an allergic problem, hay fever for example, (1) heredity, (2) seasonal history, (3) skin tests, (4) anti-bodies, (5) induction of an attack with pollen, (6) hyposensitization or avoidance of offending substance in controlling attacks; so in the psychosomatic problem we try to establish (1) the family history (heredity and pseudo-heredity), (2) evidence for a childhood neurosis, (3) sensitivity to specific emotional factors (temporal relationship of present illness and emotionally disturbing event), especially at epochal or crucial life periods (puberty, marriage, childbirth, climacteric, etc.), (4) a specific personality structure (other evidence of neurosis or character disturbance), (5) demonstration of specific behavior on taking the history (artificial exposure to a conflict situation), (6) hyposensitization by psychotherapy or the avoidance of the provocative situation. We will not be able to establish all of these postulates in every case. For example, evidence for a childhood neurosis will often be missing—neither the patient nor his family can recall disturbed behavior or bodily dysfunction.

So far as allergy is concerned the fact that the removal of an allergen or a hyposensitization process "cures" the patient proves only that one factor has been removed and the morbid chain of events interrupted. Exactly the same reasoning can be applied to psychologic factors.

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* The term psychosomatic as used in this paper indicates: (1) a method of approach to general medical problems, the simultaneous application of physiologic and psychologic techniques to the study of illness in preparation for comprehensive medical care; (2) in a more limited sense, a disorder which can be understood only when psychologic as well as physiologic factors are taken into consideration (Halliday).

Organ Neuroses

Certain psychosomatic disorders have been spoken of as organ neuroses. From a psychologic standpoint there are different varieties of organ neuroses depending upon the severity of the underlying disorder. There are very mild disturbances of organ functioning, psychosomatic dysfunction, which are hysterical symptoms. They are spoken of as conversion phenomena, a substitute expression of an emotional tension which cannot find adequate outlet. The substitute expression is symbolic, i.e., nervous vomiting may, as part of its meaning, express unconscious disgust. There are other disorders, however, which have been referred to as vegetative neuroses. These arise not as an attempt to express an emotion but as the physiologic accompaniment of constant or recurring emotional states. Here the somatic symptoms are not as much substitute expressions of repressed emotions as they are normal physiologic accompaniments of the emotional state (Alexander[5]). They are the adjustment of the organism to definite tasks which it has to face in a danger situation. They represent a utilitarian preparation and an adaptation of the internal vegetative processes (homeostasis) to a specific type of behavior which is requested from the organism. It may then be that the chronicity of the emotional tension (plus other factors) makes the condition morbid-psychosomatic organic disease. Any vegetative nervous system disturbances, such as the allergic disorders, deserve study of the psyche as well as the soma and often will prove to be psychosomatic affections.

The emotions often exploit an organic illness and thus it is that frequently following an infectious disease or operation convalescence lingers and invalidism sets in. The explanation so often given is that the organic disease produced the neurosis whereas the actual mechanism is that the organic process has broken down the individual's defenses, regression occurs, and the individual's predisposition, determined by the personality structure, permits the neurosis to emerge.

An important subject in psychosomatic illness is the shift from somatic expression to the mental sphere. Very often in the organ neuroses the improvement of a symptom because of treatment or for any other reason may lead to mental symptoms such as mood disturbances and sometimes to manic and depressive states. An important therapeutic lesson comes out of this consideration because sometimes in psychoso-

matic illness when we get rid of symptoms by means of medicine, surgery, or manipulation, without getting at the fundamental problem—the emotional conflict—the difficulty immediately is reflected in mental symptoms.

Definitive Diagnosis

Just as in a consideration of somatic disease it is necessary to make a complete diagnosis before we can hope to apply scientific treatment so it is equally necessary in psychosomatic medicine. Hence just as in general medical teaching we have always emphasized etiologic, anatomic, and functional diagnosis, so in psychologic medicine, as pointed out by Levine,[6] it is necessary to make a clinical, dynamic, and genetic diagnosis before one can stand on safe ground in regard to psychotherapy.

The clinical diagnosis in psychosomatic medicine refers to the structural and physiologic deviations as well as to the underlying or associated psychologic disturbance. For example, in the so-called organ neurosis, we would like to know whether we are dealing with a mild personality disorder such as hysteria or a severe personality disorder such as hypochondriasis. It is important, for example, in asthma, to know whether the symptoms are on the basis of conversion hysteria or a part of the clinical picture of depression in which the mood disturbance is overshadowed by the somatic complaints. When one deals with depression there is often the threat of suicide.

Dynamic diagnosis refers to the meaning and purpose of the symptoms or behavior in terms of the particular personality and its structure. Coupled with the genetic diagnosis which is derived from the longitudinal survey of the individual life history, we are then in a position to plan comprehensive medical care.

Asthma

I am not going to attempt a complete discussion of the allergic disorders. I shall select asthma first, because it is the one field in which allergists have been very willing to concede the presence of a psychological factor, and I think it is probably the one disorder that those interested in psychosomatic medicine have had the greatest opportunity to study.

Just as the heart is sometimes referred to as the seat of emotions

and the abdomen as the sounding board of the emotions, so the lungs might be spoken of as the barometer of the emotions. Under certain circumstances we catch our breath, or we complain of smothered feelings. We have shortness of breath, which represents not dyspnea, but sighing respirations which are so common in neurotic people. We speak of a person having a load on his chest, the implication being that he could get it off by talking about his troubles. Some of these symptoms will be recognized, of course, as hysterical mechanisms, the symbolic expression of tension, or to put it another way, when people are unable to act or unable to speak about a problem, the body takes over the function of answering that problem in its own way.

To go back to a former example, if someone is vomiting and no organic cause can be found, we ask the question—What is it in his environment that the individual cannot stomach? And sometimes we get at least part of the answer. In the same way we can sometimes find the cause of a load on the chest. While it may represent a respiratory disorder, it is not in the lungs; it is in the spirit of the individual, in his feelings. In other words, feelings may be as potent as germs or allergens in causing disturbances of function.

One of the things that we will have to learn in medicine is that the things people say are sometimes just as important as the sounds that their organs make. This applies especially to bronchial asthma. In other words, if we pay more attention to what people say, we will often realize that the words they produce, representing their thought processes, are the secretions of the mental apparatus, just as the urine is the secretion of the urinary apparatus, and that both can be analyzed for diagnostic and prognostic purposes.

In this connection, I would like to quote from a paper by Binger[7] on the psychobiology of breathing, in which he pointed out that the respiratory apparatus is genetically and structurally related to the gastrointestinal tract and that in many ways they seem to function similarly. Embryologically the pulmonary respiratory apparatus develops from the hind part of the ventral wall of the head gut, and he points out that both systems are concerned with the incorporation of certain substances from the external environment, with the transport of these substances to tissue cells, and with the excretion of certain products of tissue metabolism. He indicates that the digestive and respiratory systems can be susceptible to similar derangements, such as spasms and

secretory changes, and that both may be the pathways for entrance of infectious organisms. He goes on to say that if Alexander is correct in his assumption that the gastrointestinal tract may act out certain emotional trends having to do with ingestion, retention, and elimination, it is conceivable that an organ system so closely parallel embryologically and functionally can exhibit similar responses. Indeed, Alexander and Saul[8] have analyzed respiratory tracings with special reference to psychological behavior and felt that there was suggestive evidence of a relation between intaking and eliminating tendencies, observable in the emotional life and in certain characteristics of the spirograms.

In the past twenty years or more, many observations have been made and articles written on the relation of the emotions to bronchial asthma. Neurotic trends have been recognized, but, as in other organ neuroses, very often have been held to be due to the disease rather than causally related to it. It has been noted that the sufferer from asthma often seems to make the most of his attack by drawing attention to himself and by his distress disturbing those around him to the utmost. I think many of us now recognize that this is not fundamentally responsible, but it is just a secondary gain, the utilization that the patient makes of his illness, because the psychological factors that we think are related are far deeper in the unconscious mental life and have no such thinly disguised purpose. They reveal themselves only after more patient study.

McDermott and Cobb[9] in a clinical survey of fifty cases of bronchial asthma found that thirty patients showed neurotic traits other than the asthmatic and that these were usually of a compulsive character. Rogerson and associates[10] have studied children with what they call the asthma-eczema-prurigo syndrome, and they report that out of twenty-three, no less than seventeen were very overprotected by their parents, and they raised the question as to whether the difficulties produced by the disease itself had to do with this over-protection; but they go on to say that in their cases, carefully observed, it was plain that the over-protective attitude of the parents existed prior to the onset of the asthma and had a deeper motivation, such as a need to over-compensate for not wanting the child, the fears of losing the only surviving child, and a number of other reasons.

Then there is some evidence from Rorschach studies on asthmatic patients suggesting that the personality disturbance existed before the

onset of the disorder. The Rorschach test, a projection test of the personality, has been widely used in recent years and seems to be one of the outstanding methods of personality evaluation. Schatia,[11] who analyzed the Rorschach records of forty patients suffering from bronchial asthma, confirmed an impression gained by a number of analytical workers that asthmatics tend to have compulsive personalities without evidence of phobia or compulsion. Dunbar[12] called attention to this fact in 1938, and Felix Deutsch[13] made similar observations from psychoanalytic study.

The most detailed psychological study of bronchial asthma is probably now familiar to you, the publication by French and Alexander[14] and their associates at the Institute of Psychoanalysis of Chicago who studied twenty-seven cases by means of psychoanalysis.

They observed, first of all, that the patients varied a good deal in their personality traits, but one thing that existed in common was a very dependent and infantile relationship to the mother and that an attack of asthma would often occur when the patient was exposed to a situation that threatened to estrange him from the mother. This might occur in an actual life situation, in a dream, or in relationship to the physician. The attack of asthma often occurred at the point where the defenses of the individual failed and where he was suddenly exposed to the conflict between an actual life situation and the fear of losing the mother's love. These emotions often were transferred to the physician in the course of treatment so that the patient's fear of losing the goodwill of the physician would sometimes take the place of the original fear of losing the mother's love. Over and over again, French found that these patients would become blocked just at the point where their unconscious material was leading them up to make some kind of a confession, which represented an attempt at reconciliation with the parental figure. They would be afraid to confess and instead would develop an attack of asthma. As long as the technique of winning reconciliation with the mother by means of confession was successful, the patients appeared to be protected from asthmatic attacks. When the confession got choked in the throat, so to speak, an asthmatic attack occurred in its place.

French believes that throughout the lives of patients subject to psychogenic asthma attacks, there seems to run a continuous undercurrent, more or less deeply repressed, of fear of estrangement from the mother

upon whom the patient is usually very dependent in an infantile way. The cause of this fear of estrangement is usually the patient's own forbidden impulses which he thinks will offend the mother. A device that the asthmatic patient makes extensive use of to protect himself against this danger of estrangement is confession of the disturbing impulse. If the mother or mother substitute, in this case the analyst, accepts the confession without being shocked, then all is well for a time. If the patient is too uncertain of the mother's tolerance to dare make the confession, then an asthmatic attack is likely to be precipitated.

Psychic and Allergic Factors Complementary—Finally French and his colleagues conclude that psychological and allergic factors stand in a complementary relationship to each other in the etiology of bronchial asthma; that in some cases asthma attacks may be precipitated by allergic factors alone, in others by emotional factors alone, and in still others the combination of allergic and emotional factors seems necessary to bring about an attack.

Let me cite a case to illustrate some of this material.

Mrs. K. M., a healthy appearing, young woman, was first seen in June, 1946. She reported that she had had severe asthma since early childhood and had known only a few periods of real freedom in the more than twenty years that she suffered from this disorder—during the time that she was away at school and during her two pregnancies. She also had eczema as a child, and on a few occasions since, and vasomotor rhinitis which had been quite bothersome in recent years.

The mother suffered from migraine and had asthma in her early years and a brother had hay fever. The father had died of heart disease in 1942.

The patient reported that eczema began in early infancy and that asthma occurred at about the age of two and this was confirmed by the mother. The asthma became quite severe about the age of seven and she was studied in a large hospital where allergic tests were made for the first time. These had been repeated frequently since, the last time three years ago in the course of a complete study at a well known clinic. Certain foods were denied the patient which she says she eats with impunity during the periods when she has been free of asthma.

Physical examination showed a robust young woman with no evidence of eczema, rhinitis, or asthma at the time of examination. She appeared to be in good health and no evidence of organic disease was

detected. Routine laboratory studies were negative.

She was an obedient child who was taught cleanliness early. She was brought up in a small community where the parents were in comfortable circumstances so that she enjoyed all of the material possessions that a child could want. In fact she reported that her father, a tense, nervous, and domineering person "gave gifts instead of understanding." He demanded exacting obedience and the patient resented his treatment of her as well as of the mother. The father was particularly emphatic in his instructions to the young girl regarding men so that she was constantly warned about the dangers of murder and rape. Pity for the mother, the mother's air of martyrdom, and her threats of withholding love, forced the child into a submissive attitude. She tried to make up to the mother for the way the husband treated her.

She looked to the mother for approval, was very dependent upon her and yet, as she reached later childhood, found difficulty in accepting gifts from the mother. She felt that there was something wrong about doing so and this feeling has continued to the present time. Apparently accepting a gift was an indication of the dependence against which she struggles. When she went away to school, for the first time she became free of asthma for a long period and the asthma would recur only when she returned home at vacation time. In the junior year she was assailed with doubt regarding her choice of career but was afraid to change because of the mother. In order to solve the problem she became engaged, married the following year, and immediately became pregnant "in order not to have to finish school." She was well during the pregnancy but a few weeks after the birth of the child asthma began again and she then decided to leave home and move to Philadelphia. The father objected but she left anyhow and shortly afterwards the father died of heart disease. She felt no grief, "only a slight guilty feeling"—if she had not left "this might not have happened."

Sexual intercourse, which had been painful before the birth of the child, continued unsatisfactory. She became concerned about frigidity and decided to consult a physician but again asthma interfered. Another period of hospital observation failed to help the patient and she and her husband decided to move to Florida. However she did not do well there and they ran into great difficulties with the husband trying to earn a living and take care of the sick mother and child. The patient came North to visit the mother who had remarried so that she no

longer had to worry about the mother "being alone," but the asthma continued. When her child was three she decided that having been well during pregnancy another pregnancy would be helpful. True enough she again felt completely well during the second pregnancy but again developed severe asthma shortly afterwards. Now the husband's difficulties were redoubled because he not only had to work harder to earn a living but had a very sick wife and two young children to look after. The patient went from doctor to doctor and finally one suggested that emotional factors must have something to do with her illness. For the first time she began to think of her illness from that standpoint and, recalling that during her school days she was entirely comfortable away from home and sick as soon as she got home, she concluded that her relationship to her mother must be the reason.

Now as the patient "thought the problem out for herself" she went through a very bad period. She was sleepless and despondent, had a "terrible, queer, detached feeling," as if she were in another world, suffered from dizziness, pressure on the head, blurred vision, had fears that she would lose her mind or commit suicide, and was unable to be alone with the children for fear that she might harm them. Her physician was in constant attendance and she poured out to him many of the thoughts regarding her life and her relationship to her parents, husband, and children. She spoke of resentment against the father, of her hated dependence upon her mother, and of a nameless resentment against the husband "as though she would like to throw a knife at him." At the same time she admitted that he was an extremely able person and an excellent husband. If anything "he was too good" to her. She decided that she had been escaping from life and that with the doctor's help she would learn to face things. Now her black moods came and went and at times she felt exhilarated. For the first time she was able to achieve orgasm in intercourse. She felt like a different person, as if "a new personality was emerging." She described this new person as more adult, possessing a great deal of self-confidence. For several weeks she continued in this mood and was quite free of asthma but then for reasons that she did not understand her chest tightened up again and she became as ill as before. They decided to leave Florida and on the way North by automobile she was very ill, causing the husband a great deal of distress and effort as he looked after her and the family.

After establishing herself in Philadelphia she very quickly improved

as she was encouraged to talk about her life situation. Almost her whole conversation centered on the topic of her relationship with her mother and just as she described her life up to this year as one of affection and devotion to the mother now her sentiments were just the reverse. She could find almost nothing good to say about the mother. In an interview with the mother I did not gain the impression that she was mean or malicious but it was apparent that because the mother had been deprived of love as well as material things in childhood she was determined to see that her own child was loved, protected, and denied nothing. Thus she over-protected the child, confided in her regarding her loveless marriage, and made the girl too dependent upon her by threatening her with loss of love; and these were the conclusions that the patient had reached in her self-analysis. She described the mother as an infantile person who had encouraged the child's dependence, over-protected her to satisfy the mother's emotional needs, dominated her by threatening loss of love and by her suffering and martyrdom, withheld help when it was necessary and gave gifts that were not wanted. The patient had some insight however, because she spoke of desiring a better relationship to the mother with neither the affection she had felt before nor the hostility which she now feels. The mother would call on the phone and say that she was going to visit the patient and as she would hang up the receiver the patient would develop an attack of asthma. The mother would send gifts that the patient found either impossible to accept or if she did accept them she promptly lost them. On one occasion she lost a valuable piece of jewelry and felt no remorse. This quixotic behavior was not confined to the mother, however, because on one occasion the husband gave her a $20 bill which disappeared the next instant and was found in the baby's playpen. The husband recovered it and handed it back to her, telling her to be careful, and the next moment it was gone again. This time it was found in the waste paper basket.

Her dreams dealt with frustration. She was tied, bound, in a car that could not get up a hill, or in a plane that could not reach its destination. About the time that she was trying very hard to get established in a home she had a dream of "buying a house with a fence around it. In order to get the house she had to take the fence." Using this very obvious symbol I asked her what fenced her in and her reply was her illness, her mother, and housework. She was trapped in her adult

life as well as in her childhood by the dependent relationship upon her mother which she hated but from which she could not escape. However, it seems significant, that all of the time she was freely expressing her hostility for the mother she was free from asthma except for very minor attacks occurring in direct connection with the mother's visits, presents, or phone calls. The husband observed that when the mother visits them the patient "withdraws from him, not only sexually but in every way."

Summary

A young woman suffered from asthma which began at the age of two. Eczema preceded the asthma and vasomotor rhinitis occurred in recent years. There was a family history of migraine, asthma, and hay fever. Allergic factors apparently were not important in relation to the patient's illness; psychological factors seemed to be. Such was her dependency upon the mother's love and her fear of estrangement from the mother that she married and became pregnant in order to solve a conflict in regard to finishing school rather than confess to the mother her inability to pursue a course which she no longer wanted. On another level and untouched by therapy was her disturbed relation to her father which revealed itself in her relations with her husband.

When for the first time at twenty-six it was suggested to her that emotional factors might have something to do with her asthma she went through a period of self-analysis which apparently precipitated a near-psychotic reaction. The self-analysis brought into consciousness a deeply submerged hostility to the mother so that the patient, who had previously felt only affection and respect, now could hardly abide the mother. A physical contact such as a kiss or an embrace revolted her. But in the period of conscious devotion (and repressed hostility) she suffered almost constantly from asthma while in the period in which she expressed her hostile feelings she has been comparatively well. Perhaps not entirely well because other factors, constitutional as well as psychological, also enter and moreover she cannot adequately express all of her hostility by talking. To make an adult of her would be a difficult job because of her deep infantile attachment to the mother and only prolonged psychoanalysis could hope to accomplish such a task. Since that is impossible we must get along with less and it is hoped that this may result in some permanent benefit even if "cure" is not brought about.

Migraine

Migraine lies in a territory that has been fought over by allergists and psychiatrists, both claiming it for their own, but each recognizing that there are aspects of the disease that relate to the other's approach. There is considerable agreement regarding the physiological mechanisms involved. Engel and associates[15] have made some observations in the course of aviation medicine experiments about a migraine-like syndrome complicating decompression sickness. They describe the scintillating scotomas, the focal neurological signs, and the headaches which emphasize the similarity between this syndrome and clinical migraine and suggest that the mechanisms are similar. Their studies confirm the observations of Wolff[16] on the mechanism of the neurologic prodromes of migraine and suggest the identity of the two.

The characteristics of the neurologic disturbances indicate that they originate in the cerebral cortex and probably result from spasm of cerebral arteries. Moreover, the authors discovered a high incidence of migraine-like headaches in the subjects who were susceptible to this decompression syndrome, indicating that a predisposition to this particular type of vascular reaction is an important factor. They accept the interpretation that the scotomas and other neurologic symptoms result from cortical ischemia due to spasm of intracranial arteries, but that the headaches result from dilatation of pain-sensitive cranial arteries. The headache was not associated with any changes in the electroencephalogram and that is consistent with the experience of others in the study of clinical migraine.

Apparently these mechanisms can be set in motion by psychic stress related to the personality structure of the patient with migraine. In a review of twenty-four patients studied from a psychosomatic standpoint I was impressed with the features that they shared in common—a compulsive character, an inability to express hostile impulses adequately, the high incidence of various degrees of impotence among the men and frigidity in the women, and the patient's willingness to blame his headache upon the bowel, the sinuses, or "something he et". To the last I usually answer "no, it is probably something you met" but because of this association to the gastrointestinal tract and the willingness of the patient to blame the headache upon a disturbance in bowel function he usually becomes addicted to the laxative or enema habit

while, for the same reasons, his physician endorses the allergic approach. Blaming the disorder on the sinuses is also very common, so common in fact, that the patient often uses the expression "I've got my sinus again" rather than to say that he has a headache.

The role of repressed hostility has also been stressed in hypertension and perhaps the fact that migraine occurs so frequently in hypertensives indicates this common denominator in their psychopathology.

A careful study of migraine by means of the Rorschach method, using control groups (Ross and McNaughton[17]) fails to confirm the clinical finding of repressed rage but does corroborate the personality features, namely; persistence toward success, difficulty in sexual adjustment, perfectionism, conventionality, intolerance, and in general, obsessive-compulsive features. They found that these personality features are associated with migraine to a greater degree than would be accounted for by chance.

Fromm-Reichmann[18] in a psychoanalytical study of eight patients with migraine found that the individual cannot express his anger properly, and he then takes it out on himself, so to speak, in an attack of migraine. It is almost as though he would punish himself for the destruction that he would like to visit upon another. The necessity for the degree of repression of hostile feelings and why the head is chosen as the organ to express the disturbance also interested her but I shall not take the time to discuss her views on these subjects.

The following case suggests this mechanism and corresponds to the postulates that I mentioned earlier for a psychosomatic affection.

A Jewess of twenty-eight, was raised in an orthodox home, where, however, in her own words, religion was applied with a light hand. She married a man who observed the orthodox tenets of his religion with an almost fanatical zeal. Although migraine headaches antedated the marriage they now become very severe and occurred quite regularly on Saturdays.

The patient came from a family that was highly intellectual, but quite psychopathic. She considered herself scholastically the black sheep, although she was really a very intelligent girl, self-educated, and had accomplished a great deal for herself. The mother had died of bronchial asthma about the time that the patient's headaches began. The patient attributed her headaches to sinus trouble.

The patient stated that she had had a sense of relief when the

mother died because the mother had been so ill with the asthma. Then the father remarried a rather ignorant woman (these were the patient's words) in sharp contrast to the patient's own mother, and apparently this had something to do with the patient's determination to get married herself.

The patient had a fear of developing asthma, because of the mother's illness, and she had also a great fear of cancer, a real cancerphobia. Just before I saw her, she had had an anxiety attack and since then she had been nervous, weak, and depressed.

She had been studied by allergists who, because of the occurrence of the headache on Saturday, suggested that something in the orthodox food rituals might be related to the attacks. So she was subjected to skin tests, and to a number of elimination diets which, however, failed to help her.

Much of her conversation had to do with her husband. She said that his whole life was religion; that his fanatical traits are especially marked on Saturday, the sabbath day; that he rushes her to get ready for that day and that she doesn't like to be rushed. There must not be a spot of dust for the holy sabbath. On one occasion, he spoke to her about the fact that he couldn't love a woman who didn't see eye to eye with him on his religion. She resented this, saying "His love of religion is enslaving me. If love is based on ritualistic observance, he can keep it. He says he can only love me if I give up my rebellious ways. I want to worship in my own way. He forces his religion on me and he won't compromise."

In additon to that fact, just when she was so irritated and fatigued by preparation for the sabbath, it was invariable on Friday nights that he asked for sexual intercourse. There is a special preparation for intercourse among those who observe the orthodox tenets, the woman has to immerse herself completely in a tub reserved for that purpose. She very much resented this preparation. She was frigid in intercourse, failed to obtain any satisfaction, and regularly on the night or the morning following she developed her attack of migraine. Before this was called to her attention she herself observed that when she was away for the summer, she didn't have migraine; in fact, she noted, "When I am away from home, I don't get migraine on Saturdays." When I pointed out her inability to express hostility directly and hence the mechanism of body language, she replied that "hers was very eloquent."

She spent the summer away from Philadelphia. On her return, after having been free of migraine, she became irritable and depressed, complained of a heavy head, spoke to me about the boredom of facing another winter, said that she felt alone even when her husband was at home.

Finally, with my persuasion she decided to get herself some employment. I suggested that she try to find something that would fall in with her husband's interests. She got a job teaching Hebrew in a rabbinical school, and derived a great deal of satisfaction from it. Her migraine improved during this period. The anxiety of the cancerphobia came up again. I discussed that with her. She said, "Yes, people sympathize with the headache, but not with anxiety. Even my husband walks around on tip-toe when I have my headache."

I cite this case to point out some of the mental mechanisms that seem to be important in migraine. I cannot deny that allergic factors may also be involved although they apparently were pretty well excluded in this case. However in other instances it is likely that psychological factors and allergic factors are complementary; that just as French stated in the study of asthma, in the one instance it may be allergic factors that bring about the attack, in another instance it may be the psychic factors, but that in so many instances one complements the other.

Neurodermatitis

We speak of the skin as we do of the eye, as an organ of expression. The skin and the eye and the hair sparkle, they glow with vitality or they are dull and lifeless. The skin, like the eye, is an important point of contact between the inner and outer worlds. Blushing, pallor, and sweating are well known skin phenomena that express behavior. But beyond this obvious relationship we would like to know whether emotional factors may be responsible for skin disorders.

I think the general opinion of dermatologists in this regard has been well expressed by Sulzberger[19] who found no proof for nervousness as causal in the production of a dermatosis. He said, "In our material, we have gained the impression that these occasional instances of nervousness were, one, purely coincidental; two, concomitant, that is, psychoneurological disturbances caused by the same factor or factors which produced the dermatologic manifestation; or, three, the entirely

comprehensive result and the normal reaction to the dermatosis and its maddening itching, loss of sleep and continuous worry about disfigurement, etc."

Continuing, Sulzberger pointed out that the older designations of neurodermite and neurodermatitis, had nothing to do with the present meaning attached to the words nervous, neurogenic or neurotic.

In this connection, I would like to cite very briefly a case that is now old in the literature but which has some keen and critical comment that I think is still pertinent. Rattner[20] reported the case of a young man who two weeks after his marriage developed an acute dermatitis on his face, neck, and upper half of the body. He had a psychoneurosis and anxiety state. It was assumed that the dermatitis was a neurogenous dermatitis. It was subsequently shown, that the acute dermatitis was excited by perfume cosmetics which his wife used.

For sixteen years this patient had had recurrent attacks of eczema, all of which were considered to be seborrheic dermatitis. Then, superimposed on this seborrheic habitus, the acute dermatitis was found to be a sensitization dermatitis. The importance, if any, of the neurogenous component, Rattner went on to say, could be estimated only by inference, whereas positive patch tests were tangible evidence that the irritant was at fault.

I had some correspondence with Dr. Rattner about this case and he said that the diagnosis, neurogenous dermatitis, was made by a dermatologist in another city, only after a thorough search for irritants had failed. Another capable dermatologist concurred in the diagnosis. The patient then consulted a neuropsychiatrist who agreed that there was a psychoneurosis, but felt that the dermatitis was a thing apart.

I won't go on with the details of correspondence, except to say that the patient eventually got himself into the predicament of going to a psychiatrist for treatment of his skin and to a dermatologist to look after his psychological disturbance.

Dr. Pusey commented about this case: "We are witnessing an intense agitation of the subject of nervous eczema. Dr. Rattner's case illustrates the pitfalls into which we are apt to get in following this lead. Here is a case which seemed made to order for the diagnosis of eczema of nervous origin. The sexual element is exaggerated, the psychic factors are all there, including, as is usually found in such cases, a readiness of the patient to accept the emotional origin of his trouble.

The background is perfect and it takes but a few bold strokes of the sympathetic artist to give a striking picture of a neurogenous or psychogenous eczema. But what do the unsentimental facts show when they are worked out? The patient is sensitized to perfume and that is what is exciting his attacks. Many similar cases of nervous or other systemic origin which have vanished into thin air when they are traced down to their local irritations must occur to everyone with a large experience in skin disease."

"The insistence of the importance of nervous factors as a cause of eczema and many other dermatoses is a backward step into the old maze of conjecture, out of which we have been trying to find our way for more than a century. Each revival of the conjecture gives us a new set of terms, but the idea remains the same. Forty years ago they were treating neurasthenia by cutting off prepuces, correcting defects of vision and removing other actual or imaginary causes of reflex irritation to cure eczema. Then they called them eczemas of reflex nervous origin. Now we are psychoanalyzing them and calling them psychogenous and neurogenous eczemas. The words are new, but it is the same old tool. The ideas are remnants of the old hippocratic humoral pathology which has obfuscated our views for twenty-five years."

"One of the chief businesses of dermatology since it has been able in the last hundred years to study more accurately the physical and chemical facts of pathology has been to show in respect to one disease after another that these diatheses are broken reeds, extremely tempting and appealing in one way or another, according to the predilections of the individuals, but in the end broken reeds. When the diathesists are confronted with a case which they would have of a nervous origin but in which it has been demonstrated that the external cause which excites the eruption and without which the eruption would not exist, they are wont to take refuge—" and now you will recognize why I am quoting this in such detail—"in the explanation that the irritant is actually only one of the causes, that their theory still holds good because there are emotional or other factors in the case which may be contributory."

"That sort of reasoning," he states, "is begging the question. The same facts apply to every pathologic condition of specific origin that can be conceived. A longer list of predisposing causes can be offered for tuberculosis. One could even get up a list of respectable causes for scabies. But these predisposing factors in themselves are not the cause

of the disease. You may emphasize them and elaborate them. You may indulge in all kinds of intellectual and physical gymnastics, but they alone are impotent; without the definite, specific cause, disease does not occur. And it may be added in the case of irritant dermatides, without the discovery of the cause, treatment is likely to be ineffective."

I won't go on with his discussion any further, because I think I have demonstrated his point of view and the point of view of a great many dermatologists today. For the sake of discussion let us take a case of fungus infection of the feet recently reported by Harris.[21] As you may know this is a condition that is often complicated by an allergic reaction.

A virile-appearing, handsome, twenty-four year old Marine had spent 210 days in naval hospitals during a two year period of service, most of this time because of recurrent fungus infection. Personality study revealed a severe anxiety neurosis manifested by a great many psychosomatic complaints, very low self-esteem, and an unmistakable feminine trend in job preference. When the data were discussed with him he admitted bisexuality. Apparently the stress of constant stimulation and threat of exposure kept him in a state of anxiety. He was given sufficient insight to understand the mechanism of his trouble: Homosexuality–anxiety state–excessive sweating of hands and feet–inability to cure the fungus infection in the presence of constant moisture.

This case of course cannot serve as an example for a complete presentation of the controversial problem because no one would contend that there is a specific relationship between this patient's personality and the fungus infection. And yet I think every one would agree that this is truly a psychosomatic problem from the standpoint that psychic and physical factors act in a complementary fashion to produce the disorder and that only by the utilization of psychological as well as physiological techniques can we understand the illness. But of course the real problem in regard to psychosomatic medicine is whether there is a specific relationship between the personality and the skin disorder and the dermatologist wants to know whether psychogenic influences can be responsible for an actual dermatosis. The following case is intended to illustrate, not prove, this proposition.

A woman of fifty had typical neurodermatitis behind the ears and the back of the neck, sometimes extending to the arms. In addition she suffered from asthma, migraine, and hypertension. The migraine

had begun in adolescence, the asthma and skin trouble had been present for perhaps fifteen years, and hypertension had been discovered in the last several years. In the beginning the migraine had been associated with menstruation which was irregular and painful. During high school she had suffered from "anemia" and had to give up school in her third year because of "fatigue." She had been free of headaches in the last several years–her physician has said "either they would wear me out or I would wear them out." But the migraine apparently was replaced by the other difficulties. The patient had given up many of her household and social activities because she was "too nervous." She slept poorly and blamed it on the irritation of the skin. She had been studied in many excellent clinics and the diagnosis of neurodermatitis was well established. Allergy studies and elimination diet had not proved helpful.

The patient had been brought up in a small mid-western town by wealthy parents. The father was a benevolent tyrant; the mother a neurotic and over-protective person. She was married at twenty-seven to an inadequate person who never made a satisfactory living. She had known him for four years but had to wait until her older sister married before she could marry. Then her marriage was disturbed by the mother's final illness and the mother's death took place a short time afterwards. The patient had had three pregnancies but only one child, a daughter now married and living in another city.

The patient had always been frigid and in the last several years the husband had become completely impotent and there was a great deal of resentment on the part of the patient. She thought that her husband was unfaithful and in an off guard moment blurted out that "she hated him." After about ten years of marriage the father died and a great deal of trouble arose in the settlement of the estate. There was a quarrel between two brothers who have not spoken since and the patient played a buffer role between them, her sympathy being with the younger brother who suffers from a heart ailment. She feels that he was cheated out of his fair share of the estate. Because they are a close-knit family living in a small community she is constantly reminded of and humiliated by the family quarrel.

She became aware of the fact that her feelings had much to do with her illness and that she had retired into herself nursing the family problems. Attention was directed to the life situation rather than just to the skin, the asthma, or the high blood pressure. In fact nothing was

done as far as the skin was concerned—it was largely ignored—while attention was centered on the main life problems of resentment against an indaequate husband, the highly charged tension of the family schism, the retirement from life's activities, preoccupation with symptoms, and the attendent neglect of personal appearance. As she learned to express her feelings and saw her problems in a somewhat different light, her attitude changed both toward her illness and toward the family situation. She was encouraged "to carry on in spite of symptoms" and this meant doing more work, such as needlework and cooking—which incidentally kept her hands busy so that she bothered her skin less. It also meant going out more socially "in spite of the appearance of the skin." Her improvement was reflected in all aspects of her personality. She became more reassured, lost twenty pounds of her excessive weight, "spruced herself up as she came out of her shell," and took up many of her former life activities. The clearing of the skin coincided with the other improvements and a check-up two years later found her in good shape even though she had returned to her former environment.

Now the question arises—are these various disorders related to one another and to the personality of the patient. Studies demonstrate that they have certain features in common—which correspond to the clinical picture of a vegetative neurosis and meet the previously discussed criteria for a psychosomatic affection. In other words there is a positive family history, evidence for childhood neurosis or psychosomatic disturbance, the personality structure of neurotic character, exacerbation at crucial life periods in connection with specific life situations, demonstration of specific behavior on exposure to a conflict situation such as may occur in a medical interview, and clearing of the skin by the hyposensitization of psychotherapy or the avoidance of the trouble by avoiding a provocative situation.

Personality Trends in Allergic Individuals

Although the work that has been done on the question of the specific relationship of personality to disease is impressive it cannot be regarded as conclusive. Nevertheless certain trends within the personality seem to favor certain disturbances.

In regard to this question Saul[22] calls attention to the fact that it is the oral form of attachment to the mother, consisting of a mixture of impulses, desires, and feelings which become interwoven with sucking

and later the eating mechanisms, that enters into psychosomatic gastrointestinal problems. Other biological mechanisms and forms of attachment to the mother seem to be important in the allergies. These are the dermal and respiratory. In other words, in many persons the form of attachment to the mother as seen in fantasies, dreams, and real life is not, as in many instances of gastrointestinal disorders, strongly "oral," but consists rather in a desire for shelter. The longings are represented not by wishes to be fed and all that this can imply emotionally, but rather in wishes to be sheltered and protected. Such individuals often gravitate to modes of life which gratify such tendencies. Perhaps it could be said that, given a choice, they would prefer snug housing to good food. Here, too, can be points of weakness and fixation, to form a physiological pathway for the attachment to the mother and become interwoven with powerful feelings and longings.

When the relationship to the mother, with all of its significance to the child and later in life, unconsciously, to the adult, is threatened; or when a person is under stress, the longings for help or consolation are expressed in various combinations or forms in different persons—wanting to be fed, wanting to be carried or lead, wanting to be snuggled and sheltered, and so on, reflecting the oral, ambulatory, dermal, respiratory and other mechanisms and forms of attachment to the mother. The impulses may be gratified by personal relationships, sexual or sublimated, which reëstablish in some degree the relationship to the mother. The gratification may be sublimated (oral—eating and drinking; respiratory—talking and crying; skin—baths and massage). The impulses may be repressed so that symptoms appear when the tension disturbs organ function. Of course oral as well as dermal and respiratory trends can exist in the same individual. But Saul states that it is the person in whom the dermal and/or respiratory mechanism has some weakness, or is a point of fixation, or in whom the attachment to the mother predominately takes these forms, who seems to be predisposed to skin and respiratory allergy.

He concludes—"the dermal and respiratory mechanisms, trends, and relations to the mother are analogous to the oral ones. They are fundamental to an understanding of psychobiological functioning. Preliminary observations strongly suggest that they play a role in the skin and respiratory allergies similar to that of the oral ones in the gastrointestinal disorders."

While we cannot delimit constitutional and acquired factors in the genesis of these disorders I think we can distinguish between the physical (allergic) and psychological factors that enter into the problems. Indeed it is essential for proper management.

A recent coöperative effort at treatment by an allergist (Miller) and psychotherapist (Baruch)[23] dealt with twenty-two patients, seven of whom were children. In all cases the history of classical allergic symptoms was confirmed by positive skin reactions to various allergens. Aware that clinical allergy must be practiced with constant awareness that psychogenic factors influence physical results, the authors utilized individual and group psychotherapy in dealing with their patients. Repeatedly they observed that the fluctuation of symptoms paralleled the degree of freedom with which a patient expressed his feelings and that the intellectual appreciation of the dynamics involved was not important to the patient. Marked improvement was observed in nineteen of the twenty-two allergic patients. They feel that allergic symptoms express hostility, mask a feeling of guilt or anxiety, and at the same time represent attempts to gain sympathy.

Summary and Conclusions

The allergic and the neurotic populations are so large that they must overlap. If for no other reason, therefore, these disorders will exist in the same individual. But, in addition, personality studies suggest a more intimate connection—a specific relationship between neurotic character structure and allergic disorder—possibly representing parallel manifestations of the same basic fault, the one discharging on the level of psychic representation through thoughts and feelings and the other on the physiological level by means of disturbances in organ functioning.

Psychosomatic study of an allergic problem, therefore, utilizes separate techniques—psychological and physiological—applied simultaneously; and diagnosis must be established not simply by exclusion or evaluation of physical factors but with additional positive evidence of personality disorder meeting certain psychosomatic postulates. This will demonstrate that in a given case physical and psychological factors act in a complementary fashion to produce the disorder—in one instance specific physical factors may predominate, in another instance specific emotional factors. The latter seem to be determined by certain trends within the personality—just as oral attachments seem to determine

gastrointestinal disorders so do respiratory and dermal attachments (to the mother) apparently determine respiratory and dermal allergic manifestations (Saul).

Migraine seems more closely related to the character structure and personality trends observed in patients with essential hypertension, having to do with the amount and disposition of hostile impulses.

The allergic disorders seem to fall for the most part into the group of organ neuroses that can be termed vegetative (Alexander), representing early and profound deviations of personality development. What role the constitution may play cannot be determined—no methods are available to delimit constitutional and acquired factors. One can, however, evaluate physical and psychological factors and proper management depends on such evaluation. Then psychotherapy plus the allergic approach will mean better treatment for the individual with an allergic disorder.

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THE EFFECT OF TRAUMA AND STRAIN ON THE PRODUCTION AND AGGRAVATION OF HEART DISEASE*

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THE world is at present recovering from its greatest traumatic experience. This, perhaps, justifies a reëxamination and review of the knowledge of the effects of trauma and strain on the circulation, the more so since the war has produced new observations in this field. In recent years the subject has often been debated in the manner of a dispute with legalistic implications rather than objectively. This is inevitable where scientific proof is lacking and the possibilities of litigious heart disease are present, as in personal injury, industrial accident, and compensation cases.

Trauma is defined as an injury or wound. Such injury may vary from the damage of such shocking disasters as airplane crashes and atomic bombing to the minor entanglements of automobile fenders or the malign effects of overeating. But also this concept of trauma includes such influences as the inhalation of toxic gases and psychic injury without physical impact.

Strain, similarly, may be of short or long duration, severe, mild, usual or unusual, physical or mental.

In general, one may conclude that, in the category of harmful influences we are considering, the injury is so produced that some person, external agent, or set of circumstances to which the victim was forced to react, could be held responsible, responsible in a causative, not a legal, sense. This is in contrast with the effects of bacteria, metabolic products, or neoplastic or degenerative processes.

The literature on the subject has become extensive. One need mention only the reviews of Bright and Beck,[1] Master and his associates,[2] Boas,[3] Willius,[4] Sigler,[5] Barber,[6] Riseman and Smith,[7] Arenberg,[8] Gilbert,[9] Parsonnet and Bernstein,[10] French and Dock,[11] Blumgart,[12] Pater-

* Presented January 11, 1946 before the New York Heart Association.

son,[13] Brahdy and Kahn,[14] Fitzhugh and Hamilton,[15] and White and Glendy.[16] At least seventy-five pertinent references may be found in the past six years.

The war has drawn attention to such syndromes as the effect of blast concussion, airplane accidents, and fatal coronary artery disease in young men. Autopsy correlation has been of convincing value. None of us who has served in the Army or Navy can retain a dogmatism about the subject based on civilian experience alone.

Types of injury. It is customary to divide the types of injury to the heart due to trauma and strain into (1) Direct penetrating. (2) Direct non-penetrating. (3) Remote injury to other parts of the body or the effects of unusual physical exertion. (4) Psychic injury.

Only the first two may ordinarily injure a normal heart, but a severe general injury may rupture the heart, and strenuous physical effort may rupture a normal aortic valve or normal chordae tendineae of the mitral valve. That all of these injuries may aggravate a preëxisting cardiac disease may be admitted. Only long experience and a judicial mind can allocate the degree of damage due to the chronic process and the added disability attributable to the injury. Furthermore, the probabilities of injury and the type of injury due to trauma or strain will vary with etiologic type of the underlying heart disease. Aortic aneurysm, for example, would be liable to a rupture from strain, whereas auricular fibrillation would be more likely to ensue with mitral stenosis.

While interesting data from combat experience in this war have been added to our knowledge of *direct penetrating injury* of the heart the mechanisms are simple and obvious and will not be discussed here. Recovery with foreign bodies in the heart wall may be miraculous. The escape of the heart from injury may also be surprising. The author saw in consultation some thirty-five patients, aboard a hospital ship off Tarawa, who had been shot through the chest, with foreign bodies often lodging in the lung or mediastinum, without cardiac injury. An obvious conclusion would be that those shot through the heart or great vessels did not reach the ship.

Direct, non-penetrating, non-fatal injuries of the heart are probably commoner than previously thought by most observers. The work of Beck and others has emphasized the syndrome of the "steering wheel" injury with compression of the heart.

Arenberg noted that, in two hundred and fifty cases of non-pene-

trating chest injuries, the worst effects were in subjects with elastic chest walls, and that more damage occurred in those without rib fractures. This confirmed Sigler's[5] findings that the factors influencing the effects of trauma to the heart were (1) the flexibility of the chest, (2) the presence of underlying coronary disease, and (3) the psychoneurotic tendency or vagosympathetic imbalance of the individual.

In severe blows to the chest or in cases where the patient falls striking the thorax, it may be difficult to localize the area of the trauma. Butterworth and Poindexter,[17] however, found that the chest blows received by thirty-five Golden Gloves boxers produced no significant electrocardiographic changes in tracings recorded directly before and after the bouts. This would seem consistent with the age and normal cardiac condition of the contestants and the degree of trauma. What might happen to previously damaged hearts might well be different. We have seen one boxer, a negro, who developed tuberculous pericarditis following a precordial blow. Such a case belongs in the field of trauma and tuberculosis, rather than trauma and cardiac disease.

Certainly minor myocardial contusions may occur with relatively insignificant chest injuries, but, for proof, one must demand definite clinical signs or significant electrocardiographic changes returning to normal with recovery. What are significant electrocardiographic changes should be interpreted with extreme caution and appreciation of the normal variants of ST and T segments, especially in lead III and the chest leads, related to change of position, anoxia, and emotion. T wave inversion in leads I and II or ST segment displacements of over 2 mm. or intraventricular or A-V block are evidence of myocardial damage.

It is my opinion that failure to diagnose an occasional, slight myocardial contusion is an error more justifiable than the apprehensive opinion that minor electrocardiographic changes and effort syndrome symptoms commonly mean cardiac damage, with the resultant fixation of cardiac neurosis.

It is clear that each case must be individualized, but in the normal heart non-penetrating, non-fatal, non-shocking chest trauma may cause the following conditions: 1) Pericarditis—tuberculous, acute fibrinous, serous or hemorrhagic, or rupture of the pericardium. 2) Heart block—auriculo-ventricular, intraventicular (?). 3) Abnormal rhythms—premature beats, auricular paroxysmal tachycardia, auricular flutter, auricular fibrillation and possibly ventricular fibrillation. 4) T wave and ST

segment abnormalities. 5) Ruptured aortic valve or detachment of chordae tendineae. 6) Congestive failure from myocardial contusion; myocardial laceration with immediate or delayed rupture. 7) Angina pectoris. 8) Coronary occlusion.

Remote trauma, not producing shock, or sudden severe exertion, appears effective in these conditions: 1) Subacute bacterial endocarditis from dental extraction or from an injury which disturbs the immunity process. 2) Rheumatic fever may rarely be reactivated by an injury or surgical operation. 3) Auricular fibrillation may be induced by severe effort, injury, or electric shock. 4) Ventricular tachycardia may be produced by inhalation of chlorinated hydrocarbons–tetrachlorethylene, carbon tetrachloride, chloroform, ethyl chloride, and cyclopropane.[18] 5) The anoxia of carbon monoxide inhalation may produce myocardial necrosis and precipitate anginal or congestive failure in those with coronary artery disease. 6) Normal heart valves may be ruptured by exertion such as cranking a car, and normal chordae by an effort like rowing. Other coronary effects will be considered later.

Since these conditions have been observed and reported with adequate necropsy study, it should lead us to consider the possibility of such cardiac injury in all traumatic cases. Moreover, such patients should be kept under observation for at least two weeks with the realization that in a rare case the final effects of trauma may not appear for weeks or months, as in post-traumatic adhesive pericarditis.

The minimal evidence for injury to the heart when not otherwise explained should be one or more of these findings: 1) significant electrocardiographic changes, 2) cardiac enlargement unexplained by preexistant cardiac disease or hypertension, 3) abnormal rhythms, not including premature beats unless supported by other electrocardiographic changes, 4) pericardial friction rub or cardiac tamponade, 5) aortic diastolic or loud mitral systolic murmurs with signs of congestive failure, 6) congestive failure immediately precipitated by trauma or strain, 7) angina pectoris or myocardial infarction starting within twenty-four hours of the trauma if accompanied by distress at the time of the incident. If the injury is severe, such conditions starting at any time during convalescence can reasonably be attributed to the traumatic event.

Rarely, cardiac damage eventually fatal may not reveal itself for several hours, or even days, and in the interim the individual goes about

his business without disabling symptoms. Such has occurred in myocardial contusion leading to delayed rupture.

I have reserved for more detailed consideration the most troublesome relationship in this field, that between *coronary artery disease and trauma or strain.*

It is a peculiar fact that most everyone will agree that a man with coronary artery disease may die from sudden exertion, but there appear to be two schools of thought when doctors consider those who do not quite die from myocardial infarction accompanying or following exertion. One group believes that coronary occlusion arrives unrelated to any outside influence in the relentless process of coronary narrowing from atheroma. The other school is ever on the alert to discover in the patient's history some unusual exertion or strain which precipitated the occlusion. There is, it is true, an intermediate point of view which holds that myocardial infarction of lesser degree, subendocardial in situation, is produced by the relative ischemia of a reduced coronary flow in an atherosclerotic vessel plus added myocardial demands, the syndrome of coronary insufficiency.

Statistics have been published to show that coronary occlusion bears no relationship to effort, trauma, strain, or occupation, since no more attacks occur during the active hours of the day than would be proportionately predicted, and that many attacks occur at rest or during sleep. Other figures, while admitting the frequency of coronary occlusion during inactivity, point out that this does not preclude the possibility that a given unusual exertion may precipitate such an occlusion prematurely.

These differences are not based on a confusion of terminology since it is recognized that coronary occlusion, if slowly produced, may not cause myocardial infarction, and that conversely, infarction of the myocardium may occur from coronary insufficiency without occlusion.

The main dispute surrounds the analysis of a typical case of a patient with acute myocardial infarction with characteristic electrocardiographic changes, and survival, who had engaged in an unusual effort, or been subjected to unusual strain, directly preceding the onset of his attack. Was the attack fortuitous and coincidental or causally related to the stimulus?

Let us consider the mechanisms suggested by different authors for the production of myocardial infarct, with or without coronary occlu-

sion. Let us, furthermore, grant that underlying coronary disease is always present in these cases of myocardial infarction excepting the extremely rare direct injury to a traumatized coronary artery. In all these mechanisms a relative, or absolute, ischemia and anoxia of the heart muscle is produced.

I. *Lowered coronary blood pressure and reduced coronary flow resulting in thrombosis.* (a) Normal resting pressure for the given subject during sleep. (b) Reduced systemic blood pressure in shock. (c) Abnormal cardiac rhythms—tachycardia or bradycardia. (d) Reduced blood pressure by hemorrhage. (e) Lowered blood pressure occurring after exertion. (f) Lowered blood pressure in dehydration. (g) The reduction of cardiac output during forced expiration of effort, with closed glottis (Valsalva effect), producing coronary insufficiency.[19]

II. *Elevated coronary pressure.* (a) A rise in blood pressure from exertion, pain, or emotion results in rupture of abnormal intimal capillaries in the coronary walls. This may be favored by local anoxic effects with softening of atheromatous deposits beneath the intima. The hemorrhage may rapidly or slowly produce a subintimal projection impinging upon the opposite wall of the coronary vessel occluding the lumen, or it may rupture into the coronary vessel furnishing a raw surface for the deposition of thrombus. (b) A similar rise in blood pressure may rupture an atheromatous abscess through the intima and dislodge an atherosclerotic plaque or the contents of the abscess as material for distal occlusion of the vessel, or also produce a raw surface as a base for thrombus formation.

III. *Miscellaneous mechanisms of anoxia.* (a) Reflex coronary spasm, especially the gastro-coronary reflex occurring after meals or with hiatus hernia, gall bladder disease and other intestinal pathology. (b) The relative ischemia of effort whereby the coronary supply is inadequate to prevent myocardial necrosis in the presence of increased demands. (c) The anoxemia of anemia. (d) The anoxemia of carbon monoxide poisoning. (e) Chronic excitement of the vagus (recently disproved). (f) Inhibition of sympathetic tone as occurs in experimental animals by stimulation of the nasal mucous membrane and may occur in man by the inhalation of cold air.[9]

IV. *Other possible mechanisms.* (a) Direct mechanical injury of a coronary artery, causing spasm or hemorrhage. (b) Vitamin deficiency. (c) The effect of trauma is more severe in experimental animals under

the influence of digitalis, thyroxin, or epinephrin.

With all these possibilities one might feel indeed that any condition which disturbs the "steady state" of a person with coronary artery disease might precipitate coronary insufficiency or even coronary occlusion. How often any one of these is effective cannot be told, cannot even be suspected without much more autopsy study, nor will that answer the question of just what happens in those who survive myocardial infarction. Why then should we not be content to fall back on an appeal to coincidence, since all we can ever say is that it "might" have happened thus and so in a given case, but we cannot prove in this case that it "did" happen in this fashion?

One day last December the driver of a pick-up truck stalled his car in a snowdrift in Boston. Two men offered to push him out and one of these three men died. It seemed like a clear case of unusual physical exertion and latent coronary disease. But the man who died was the driver sitting behind the wheel of his truck and not one of the men pushing it. Was this coincidence or could we discover that the driver actually had tried to shovel the car out, that he had wrestled with the gears and steering wheel, or that the cold and the excitement of the episode were actually factors in his death? Until the whole story of such a case is known in finest detail, should we attempt a surmise? Thus it is of value to study the antecedent activities, the actual effort or strain, and the resulting condition of every patient with myocardial infarct.

In 1933 Fitzhugh and Hamilton[15] became convinced that acute coronary closure and fatal angina pectoris might be deferred in patients with known coronary disease if attention was paid to warning such patients to avoid "departures from ordinary living" or what, to borrow a term of biophysics, I have called a "steady state." They collected one hundred cases in which the following conditions appeared operative in precipitating the occlusion, or what Chief Justice Rugg called "acceleration of previously existing heart disease to a mortal end sooner than otherwise it would have come," which, incidentally, is a crucial phrase. (1) Prolonged activity and fatigue. (2) Persistence in activities that had repeatedly caused angina. (3) Travel. (4) Emotional strain. (5) Alcoholic excess. (6) Starving. (7) Medication, thyroid and bromides. (8) Sexual excess. (9) Straining at stool. (10) Gorging.

Other activities associated with myocardial infarction, including those reported by Boas are: (1) During heart failure. (2) With shock.

(3) With pulmonary embolism. (4) Acute hemorrhage. (5) Acute infection. (6) Marked tachycardia or bradycardia. (7) Hypertensive crises. (8) Chest injury. (9) Exertion—sudden, severe. (10) Allergy. (11) Cold. (12) Insulin shock and hypoglycemia. (13) Electric shock. (14) Excessive heat and humidity.

A patient in this category whom I recently examined was a sailor in his early thirties who arose one afternoon feeling well, shaved, and went to breakfast. He was suddenly stricken by the sensation of a blow in the mid-sternum as if "a man had hit me and pulled his arm away and left his fist in my chest." He survived a typical myocardial infarction. Prior to this attack he was free from all symptoms, but it appeared that he was on two weeks leave and had been drinking heavily during this time, accounting for his afternoon breakfast. In spite of what some may think this is "a departure from ordinary living" in the Navy.

Although one may remain sceptical of the causal relationship in some reported cases, the experience of World War II shows that subjecting men to exertion to which they are unaccustomed and *which they must perform in the face of severe fatigue,* has precipitated coronary failure, occlusion, and sudden death.

The reports of French and Dock[11] and of Blumgart[12] are very convincing. In the former series vigorous effort and early morning chores were the apparent cause in over fifty per cent of eight fatal cases of uncomplicated coronary disease.

In Blumgart's series of eleven cases, 30-56 years old, effort was considered the precipitant. His conclusions are notably sound: "The relationship of effort to a given attack of coronary occlusion may be certain, probable, suggestive, or improbable, or non-existent. The relation is definite if these criteria are satisfied: (1) The development and increase of cardiac symptoms such as pain or substernal distress during or immediately following unusual effort. (2) The continuation of the symptoms after cessation of effort. (3) The presence of the clinical signs and symptoms of acute myocardial infarction. (4) The development of the characteristic electrocardiographic patterns of acute anterior, posterior, or lateral wall myocardial infarction."

In 1941 Westinghouse and I[20] reported seventy-seven attacks of sudden occlusion of peripheral arteries, including the retinal arteries, by the mechanisms of embolism, thrombosis, and endarteritis in ambulatory, apparently well, individuals. These occlusions occurred four-

teen times as commonly when the person was at complete rest than when engaged in the ordinary exertions of life.

I confess that the situation in the peripheral arteries may be different from that in the heart, but it does seem as if a relatively reduced blood flow was more conducive to thrombosis than a very active one. However, a very few of these episodes occurred during severe exertion.

Willius,[4] however, has gone the whole way in one direction. Since, as he says, coronary thrombosis never occurs in a normal artery, strains in the past in the case of the afflicted individual did not produce occlusion, which occurs more often in the sedentary and at rest, then effort bears no relationship to coronary thrombosis. From a compensation viewpoint based on the emphasis on a precipitating event, he says, "The practice of genteel extortion has become so widespread as to constitute an alarming scheme of sanctioned fraudulence."

This, I feel, is a limited peacetime attitude, and too selective, but also too often it is true.

It is quite obvious that the restraining influence against soft-heartedness on our part in compensation cases is the possibility of malingering or traumatic neurosis. For that reason the symptoms of precordial pain, dizziness, faintness, sweating, sighing, and palpitation cannot be accepted alone as showing injury to the heart. Let the defense be psychiatric and not cardiologic.

But by all means let us be objective to the extent of refusing categorical answers. Let us emulate Dr. Jelley on the witness stand. He was once presented by the lawyer with the longest hypothetical question on record.[21] It was twenty thousand words and took three hours to deliver. At the end of it Dr. Jelley's answer was, "I don't know."

No matter how hard we try the legal methods may defeat our best intentions. Though we may see no relationship between a given incident and the aggravation of preëxisting cardiac disease, a lawyer may confound one by replying "then you think it is good for a man with heart disease to fall down on an icy sidewalk?"

There remains consideration of *emotional trauma.* In the main it is a good legal principle that nervous strain cannot be considered a compensable injury if the emotional trauma was of such a sort as not to be expected to harm a normal individual. Otherwise might we not all be liable for fatally irritating a man with angina?

Emotion may speed the heart and raise the blood pressure, but while

the released adrenalin acts as a coronary dilator, this beneficial effect may be negated, in the individual with coronary narrowing, by the concomitant increase in cardiac work due to the emotional discharge. Pain, induced by constriction of a limb or the cranium, will produce changes in the electrocardiogram in subjects with coronary disease.[22] The T waves may increase or decrease in amplitude, an abnormal tracing become normal, and vice versa. Occasionally this appears to be true in normal subjects.

These findings are difficult to evaluate, but show that something does take place in the coronary circuit under painful stimuli usually with increase in blood pressure, but just how this is mediated is not clear. Graybiel has shown that the startle reaction produced by firing a gun behind a normal subject can cause, in the electrocordiogram, bundle branch block, inverted T waves, and displacement of the pacemaker. One should, therefore, be reluctant to interpret too much significance into electrocardiograms purporting to indicate coronary injury from emotional stress.

The delayed appearance of myocardial infarction following a single emotional strain would seem highly unlikely. But the control of emotions may well prolong the life of a person suffering from coronary disease. One of Hamilton's patients, "a gentleman of the old school, famed for his easily aroused ire, found at the age of seventy that indulgence in bad temper caused him angina. He succeeded completely in controlling his disposition and lived for ten years in a forced good humor."

The cardiac responses effecting sudden death from exertion or emotion are not all known. Ventricular fibrillation or standstill appear to be two of them. It is hardest of all to prove the mechanism of unexpected death in apparently normal young individuals with normal hearts at necropsy.

Raab[23] has reported the autopsy findings in a young man who died during a game in whom there was a very high concentration of epinephrin bodies in the heart muscle as the only abnormal finding.

One of my pathologist friends said recently that there is no reason why a normal person could not faint to death if he were propped up during syncope. Such seems to have occurred in an exhausted soldier who returned from the battlefield, ate a heavy meal, and fell asleep in sitting position against a wall.

Finally, from a practical experience in industry what may one conclude about the *danger of employing men with latent coronary disease?* I will quote only one such report. In twenty years the Eastman Kodak Company has had only one case of a death claim for aggravation of existing heart disease. At autopsy this man was found to have a syphilitic aorta with a moderate back strain as the cause of the claim.

One hundred employees died of coronary thrombosis in the years 1921-1940. In only twelve did symptoms begin at work and in only one case was there a question of aggravation by work activity. Crain,[24] who reports this experience, states that a good preëmployment physical examination defining the disability is as good as a waiver of disability in cases of compensation.

In *summary* let me emphasize what seems necessary to further the study of this ramified subject of trauma and the heart. The desiderata in every case are these: (1) An honest history, taken as soon as feasible after the event. (2) The absence of present or impending litigation. (3) The condition of the patient prior to trauma or strain. (4) The antecedent activities of the patient for at least a week before. (5) The customary physical and emotional habits of the subject. (6) The exact degree of injury or strain. (7) The history immediately after the episode. (8) The bridging of symptoms or "intercalary period." (9) The departure of the patient from his normal equilibrium following the episode. (10) The objective evidence of cardiac injury—change in heart size or function, pericardial or endocardial variants, electrocardiograms. (11) The assessment of the neurotic component. (12) The autopsy findings or the final recovery state.

Numerically, after all, this group of cases is a small part of cardiology. I do not wish to over-accent the role of trauma or strain in the production or aggravation of heart disease, since such are the exceptional and not the usual factors. Infection is vastly more important.

But, in all cases, let me plead for an open mind, full experience, and the rule of reason. This should not be a battle of conflicting authorities in which "God fights on the side of the heaviest artillery" unless such artillery be the truth, as we know it, and not as we imagine it.

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THE EXTRA-RENAL SEQUEL TO EXPERIMENTAL RENAL HYPERTENSION*

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THE recently revived interest in the study of experimental hypertension has been largely directed toward understanding the etiology of hypertension as seen in man, especially essential hypertension, and toward producing this disease experimentally for further study. Although neither of these intentions has been crowned with direct success, our understanding of the circulation, both normal and hypertensive, has been greatly increased by these studies, and particularly those initiated by Goldblatt's[1] work on clamping the renal artery. So it seems worthwhile to consider experimental hypertension as a field of study in itself and to review some of the observations in an attempt to evaluate changing concepts in the field.

EXPERIMENTAL HYPERTENSION OF CEREBRAL ORIGIN

Since the cerebral cortex is dependent upon an immediate blood supply, and the carotid and vertebral arterial systems seem specially designed to guard it, one might expect that an interference with the proper delivery of blood to the brain would produce a compensatory rise in blood pressure. That this is not a simple arrangement is clear since intracranial vascular disease usually does not produce chronic arterial hypertension unless there is an accompanying change in intracranial pressure.

This question has been approached experimentally by Nowak and his colleagues[2] who found that successive ligation of the various cerebral arteries is sometimes followed by chronic hypertension, but Blalock and Levy[3] produced only temporary hypertension by this means.

Dixon and Heller[4] using dogs, and Griffith, Jeffers and Lindauer[5] using rats, produced sustained hypertension by raising the intracranial pressure by the intracisternal injection of kaolin. Griffith and Roberts[6]

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found about four-fifths of rats or dogs so injected became hypertensive. The inflammatory reaction and leukocytosis resulting from these injections had usually begun to subside before the hypertension appeared. This observation is in contrast to the prompt rise of blood pressure which occurs in man as the intracranial pressure rises. Reports indicate somewhat variable results with this method; Blalock in his review[7] expresses the belief "that this type of hypertension cannot be produced with great regularity and that the elevation of blood pressure is usually not persistent." At the present time it may be said that there is no clear indication that studies of cerebral ischemia by arterial ligation or the injection of kaolin have shed much light on the etiology of human hypertension except that which follows injuries, tumors, and other causes of raised intracranial pressure. Nor has there been a sufficiently varied or extensive study made of animals hypertensive from these causes to add considerably to our knowledge of the behavior of organisms as a whole, or of various organs within them, when subjected to a period of raised blood pressure.

At this time a careful experimental study of cerebrospinal fluid pressure in animals with Goldblatt hypertension might be fruitful.

Hypertension From Vitamin D and Desoxycorticosterone

The association of elevated systolic blood pressure with raised blood cholesterol and arteriosclerosis led to the experiments in which Appelrot[8] studied the possible hypertension-producing effect of Vitamin D_2. The data are insufficient to add much to our knowledge of hypertensive processes; but Handovsky[9] reports that dogs whose blood pressure was maintained at hypertensive levels for eighty days during administration of Vitamin D_2 did not continue to have hypertension after the drug was discontinued.

The elevation of blood pressure arising from injection of desoxycorticosterone is at present difficult to interpret. Other aspects of the physiology of various steroids of biological importance are at present undergoing intensive study all over the world and it seems likely that the part played by these substances in cardiovascular disturbances will be somewhat clarified shortly.

Buffer Nerve Hypertension

We may now turn to hypertension produced by experiments on

buffer nerves, some of which are already applicable to our understanding of the hypertensive process.

The study of hypertension produced by denervation of the cardio-aortic and carotid vasosensory zones has been pioneered by Heymans but this method has been used enough in other laboratories so that the essential facts are well known and are matters of fair agreement.

The pressure-sensitive nerve endings in the left ventricle, the arch of the aorta, and the carotid sinus are continuously sending afferent impulses to the medulla tending toward the reflex lowering of blood pressure. Bronk and Stella[10] have shown that each systole produces an increase in the frequency of afferent impulses from these regions and therefore tends to produce a vascular relaxation at the peak of systole. This work shows the rapidity of that reaction, its sensitivity, and the fact that it is normally operating in the pressure ranges which occur in the resting physiological state. The efferent pathways for these depressor effects are widespread but probably include diminution of sympathetically maintained vascular tone particularly in the splanchnic area.

Denervation of these vasosensory zones in acute experiments results in an immediate rise of blood pressure and cardiac acceleration, and Heymans and others have shown this to be largely due to a rise in sympathetic tone.

Chronic hypertension has also been produced by the denervation of these zones. For anatomical reasons the operation is difficult and uncertain particularly since the aortic depressor fibers in most species accompany the vago-sympathetic trunk and are difficult to isolate and divide. Such extensive surgery as that involved in denervating both aortic depressor zones and both carotid depressor zones is commonly attempted in a multi-stage operation. At best the final stage results in such profound cardiovascular disturbances as to endanger life immediately; and accidental interruption of vagus efferent fibers at the site of operation is liable to produce serious disturbances in the respiratory and digestive systems which may kill the animal later.

These reasons may account for the discrepancy between the pupils of Heymans, who after considerable experience are usually able to produce satisfactory chronic hypertension, and others who have commonly produced only moderate and temporary hypertension and so have been inclined to abandon the procedure before acquiring extensive

experience with it.

If the animal with chronic hypertension of this kind is sympathectomized completely, its blood pressure is restored to normal (Heymans[11]). There is no agreement (Nowak and Walker[2]) as to whether hypertension develops if the section of the moderator nerves is made originally in a sympathectomized animal. Grimson, Bouckaert and Heymans report[12] three animals in which they first performed splanchnic sympathectomy, complete except that the renal nerve supply was left intact. Next, they raised the sympathetic tone by denervation of the cardio-aortic and carotid zones. In these three animals the rise in blood pressure which ensued was believed to be due to renal vasoconstriction causing renal ischemia and thus hypertension of the Goldblatt type. These animals were later subjected to denervation of the kidneys and the blood pressure returned to normal. This type of study which involves three major operations: splanchnicectomy, moderator nerve section, and renal denervation, and requires repeated blood pressure measurements, is evidently difficult. This is a sufficient justification for the limited number of experiments reported. Unfortunately, in this experiment, the assumption is implied that the mechanism of hypertension once established is not subject to change; and therefore Heymans did not investigate the effect of varying the time interval between the establishment of the hypertension and its "cure" by renal denervation. An assurance that such animals would be cured of their hypertension by renal denervation even after a period of many months' or years' hypertension would be valuable in the consideration of the effects of prolonged elevation of blood pressure on the organism as a whole. This is a point of importance which will be discussed more fully in connection with chronic renal experimental hypertension.

The foregoing discussion of experimental hypertension of non-renal origin serves to indicate some of the lines of approach to the important question: Which of the phenomena of renal hypertension may properly be attributed solely to the change in intra-arterial pressure as contrasted with those effects due specifically to the kidney?

Experimental Renal Hypertension

The essential facts about experimental renal hypertension are well enough known that they need but the briefest recapitulation here.

Elevation of systolic and diastolic blood pressure may be regularly

produced by partial occlusion of both renal arteries by a Goldblatt clamp[1] or ligature (Wilson and Byrom[13]) or by the production of perinephritis such as can be induced by wrapping the kidney in silk or cellophane (Page[14]). Hypertension can also be produced by tightly constricting the kidney with tape according to the technique of Grollman[15] or by partial ablation of kidney tissue as described by Chanutin and Ferris.[16]

Hypertension produced by these means may be of rapid onset, rapid progress and lead to death; may be of slower onset and lead to stable chronic hypertension; or may be only temporary. This depends upon the procedure employed, the presence or absence of an intact kidney, the species under observation, and probably a number of other uncertain factors. These methods all produce hypertension but the mechanism is open to discussion and may now be examined.

Mechanism: It is usually believed that in such experiments the kidney liberates renin into the blood stream. This reacts with a part of the globulin fraction of the plasma (renin substrate—α_2 globulin) to form the vasoconstrictor substance, angiotonin, which is directly responsible for the hypertension by raising the peripheral resistance.

What part of the kidney produces the renin, and the mechanism of its liberation is uncertain.

Change of Mechanism During the Course of Experimental Renal Hypertension

Renin may be liberated very promptly in response to emergencies which reduce the pressure[17, 18, 19] but there is no certainty as to how long the kidney will continue to secrete renin. Probably in extreme circumstances the kidney can be exhausted in a few hours.* It has generally been assumed that the hypertension established by moderate renal artery obstruction and consequent renin liberation may continue indefinitely with no change of mechanism but since we know that renin substrate may be quickly exhausted by the rapid injection of renin or even by causing the kidney to liberate a large amount of this substance, it is becoming increasingly evident that this view must be reëxamined critically.

Renin in Circulating Blood: If renin, in fact, is responsible for ex-

* Shorr finds apparent exhaustion of his renal vaso-excitor material in four hours of hemorrhagic shock.

perimental renal hypertension, it should be possible to demonstrate its presence in the circulating blood. Attempts to do this have usually been unsuccessful but some investigators have detected renin in the blood stream of dogs shortly after the application of the clamp (Dell'Oro and Braun-Menendez[20]) and have remarked upon its apparent absence later.

Fasciolo, Houssay and Taquini*[21] found renal vein blood from hypertensive dogs to have a raised vasoconstrictor action on the perfused frog. Mason and Rozzell[22] were unable to confirm this. This disagreement was abolished when it was realized that the former had used dogs with newly established hypertension, the latter chronically hypertensive dogs.

It has been suggested that the animal may become increasingly sensitive to the pressor action of renin, and that therefore later hypertension might be sustained by immeasurably small amounts, but the experimental evidence does not allow of this being the sole explanation (Pickering[23]).

Hypertension from renin infusion[24] or from acute and severe limitation of renal vascular supply is of notably small magnitude and short duration and usually lasts only a few hours.

Renin is found during acute glomerulonephritis[25] but not in chronic hypertension in man. Pickering[26] has adduced other evidence indicating a fundamental difference in the mechanism of hypertension of acute and chronic nephritis.

Dock and Rytand[27] demonstrated the absence of vasoconstrictor substances in rats made hypertensive by partial nephrectomy, but they do not say how long the rats had been hypertensive. In view of all the other evidence for the humoral origin of renal hypertension their conclusion offers some support to the idea that more than one mechanism is involved in the course of renal hypertension in the rat.

Dock[28] made rabbits hypertensive by renal artery ligation, pithed them under anesthesia and found them to have blood pressures as low as normal animals similarly pithed. Since pithing lowered the blood pressure completely, he concluded that the hypertension was not mediated by a circulating pressor substance, but that the hypertension in these animals was due to a change in the "set" of the central nervous system, comparable, perhaps, with the change of the "set" of the thermal

* Dr. Taquini informs me that this question is now being reinvestigated by Dr. Fasciolo in Buenos Aires with recent improvements of technique. Preliminary correspondence with Dr. Fasciolo suggests that renin is demonstrable in normal and hypertensive animals, but that animals with long-standing hypertension have smaller quantities of renin circulating.

regulatory mechanism in lobar pneumonia.

Probably renin is not operating in rabbits after seven weeks of hypertension, so Dock's rabbits which had been hypertensive for "months" may be considered "chronic." This observation of Dock's that the nervous system is involved in late hypertensive rabbits and that a humoral system is not involved further supports the concept that the mechanism of experimental renal hypertension changes with time.

Pickering[23] showed that rabbits with recently induced renal hypertension have normal sensitivity to injected renin as contrasted with chronic renally hypertensive rabbits whose sensitivity is unusually great and prolonged. (These experiments are complicated by the fact that his observations were all made on animals shortly after complete nephrectomy, which of itself may sensitize to renin.)

Taggart and Drury[29] claimed that renal hypertension does not involve renin at all. They worked on rabbits with hypertension of two months' standing (quoted by Pickering) which therefore belonged in the "chronic" category. These rabbits were normally responsive to injected renin and when made tachyphylactic by repeated doses of renin their hypertension was not abolished. This suggests that endogenous renin was not causing their hypertension.

Wakerlin in 1943[30] showed that a series of injections of renin would prevent the development of renal hypertension if the injections were continued for some time after clamping the renal artery. Many dogs have been studied by him and others for the possible hypotensive effect of renin injections on hypertensive dogs. The results have been variable and in 1946 Wakerlin was at a loss to explain them.[31] An examination of his figures shows that renin given prophylactically or in the early stages of hypertension is usually effective, whereas extracts given later are variable and temporary in their effects. His results are compatible with the view that the injected renin uses up all the substrate and makes the animal unresponsive to its own renin and therefore abolishes the reno-humoral mechanism of hypertension. The variable results of injections given later may be, and indeed the published figures suggest that they are, due not to renin but to the temporary non-specific antihypertensive effect common to many tissue extracts (Chasis, Goldring, and Smith[32]).

The difficulty of an accurate interpretation of these experiments in the light of the dual mechanism hypothesis arises from the fact that the

TABLE I

COLLECTED RESULTS OF NEPHRECTOMY ON EXPERIMENTAL HYPERTENSION IN RATS

Authors	*Method of producing hypertension*	*Number*	*Result of nephrectomy*		
			No change or a rise	*Lowered, but not to normal*	*Return to normal*
Wilson and Byrom	Silver clip obstructing one renal artery	27	8	9	10
Friedman, Jarman, and Klemperer	Cellophane bag around one kidney	44	3	29	12
Patton, Page, and Ogden	Partial occlusion with silk tie of one renal artery	47	15	20	12
Totals		118	26	58	24
Per cent			22	49	29

Patton, Henry S., Page, Ernest W., and Ogden, Eric: The Results of Nephrectomy on Experimental Renal Hypertension, Surg. Gynec. & Obst., 76:493-497, 1943. By permission of Surgery, Gynecology and Obstetrics.

experiments were designed rather for another purpose than to test this hypothesis. Friedman[33] using three dogs of at least 54 days hypertension was unable to reduce the blood pressure by renin injections.

A number of workers (Harrison, Blalock and Mason;[34] Prinzmetal and Friedman[35]) have claimed that the ischemic kidney contains more renin than the normal kidney. Pickering, Prinzmetal and Kelsall[36] found this to be true for the newly hypertensive rabbit but not for the rabbit with hypertension of long standing. The change in the renin content of the kidneys and in the sensitivity to injected renin and the fact that renin can sometimes be found in the blood of the early hypertensive animal but not in the animal with long established hypertenson, suggest that there is a change of mechanism in the course of hypertension and that this is maintained at first by renin and later in some other way.

The Effect of Nephrectomy on Experimental Hypertension: Renal hypertension may be readily established in the rat by the partial ligation of only one renal artery leaving the other kidney intact. Removal of

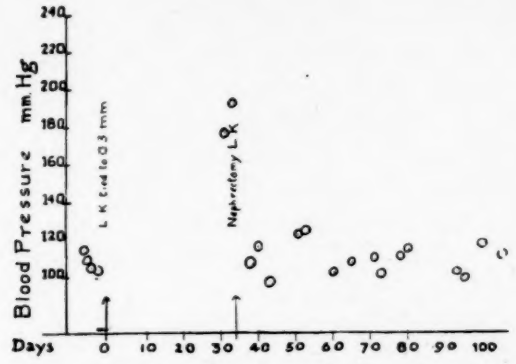

Fig. 1. Complete relief of hypertension of short duration in a rat after removal of the left kidney whose artery had been partially ligated. Right kidney intact.

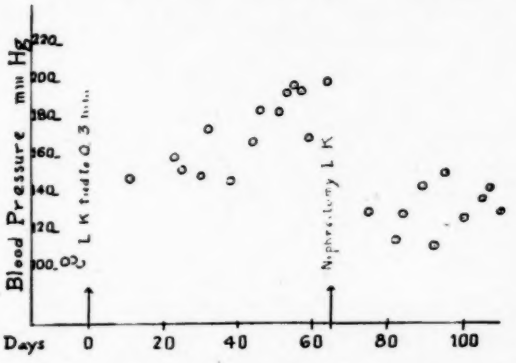

Fig. 2. Moderate improvement after hypertension of 2 months' duration.

the ligated kidney is sometimes followed by a decrease of blood pressure and sometimes not (Table I). Wilson and Byrom[18] first called attention to this and believed the *degree* of hypertension to be the determining factor.

Friedman, Jarman, and Klemperer[37] remarked on the failure of unilateral nephrectomy regularly to restore the original blood pressure in rats hypertensive from silk perinephritis. They also concluded that the restoration depends rather upon the intensity than upon the duration.

We[38] reinvestigated the effect of nephrectomy on rats made hyper-

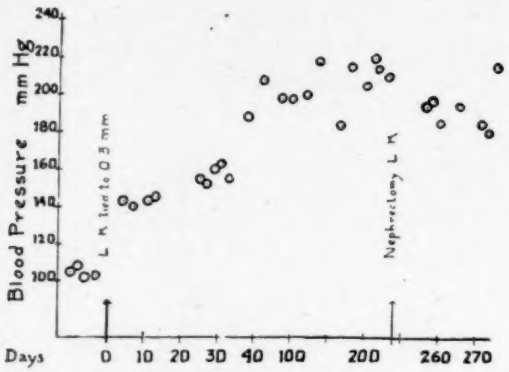

Fig. 3. No lowering of blood pressure after 7 months of hypertension. The time scale is irregular to conserve space.

TABLE II

THE RELATION OF DURATION AND SEVERITY OF HYPERTENSION TO THE EFFECTIVENESS OF NEPHRECTOMY IN RATS

Duration—wks.	*No. of animals*	*Result of nephrectomy*		
		No change or a rise	*Lowered, but not to normal*	*Return to normal*
5-10	21	2	9	10
11-45	26	13	11	2
Totals	47	15	20	12
Severity of hypertension—mm.				
140-160	26	12	9	5
161-180	11	2	6	3
181-200	7	1	3	3
Over 200	3		2	1
Totals	47	15	20	12

(Note that there is no evident relationship between the maximum blood pressure level and recovery.)

Patton, Henry S., Page, Ernest W., and Ogden, Eric: The Results of Nephrectomy on Experimental Renal Hypertension. Surg. Gynec. & Obst., 76:493, 1943. By permission of Surgery, Gynecology and Obstetrics.

tensive by partial ligation of the left renal artery. At various intervals after the hypertension was established, the left kidneys were removed. Figures 1, 2 and 3 are records selected to show that the removal of the affected kidney resulted in more or less lowering of the blood pressure according to the duration of the hypertension. Table II analyzes these experiments with respect to the effect of nephrectomy as related to both the severity and the duration of the hypertension.

These data lead to the conclusion that it is not the degree but the duration of the hypertension that counts and that the longer an animal has been hypertensive from this cause the more likely it is to remain hypertensive after the offending kidney is removed. In these experiments the obstruction of the blood supply to the kidney initiated the hypertension presumably by the renin mechanism; the maintenance of the hypertension, however, must be due to something other than the kidney which has been removed.

In view of the different interpretation of our results from the interpretations of Wilson and Byrom and of Friedman, Jarman and Klemperer, it is necessary to seek the cause of the discrepancy. Wilson and Byrom remarked upon the difficulty of interpreting their data because they cannot find a satisfactory figure to report a particular rat's blood pressure during a given period of time and so they have grouped their rats according to their judgment of their behavior. An analysis of the mean blood pressure figures given in their table does not indicate any correlation between the effect of nephrectomy and the intensity or duration of the hypertension, but a study of their grouping shows that of the 18 animals in which nephrectomy cured or substantially improved the hypertension, only three had been hypertensive for more than 10 weeks.

The data of Friedman, Jarman, and Klemperer included 44 rats of which only 4 had hypertension of seven weeks or less, fourteen of 7-10 weeks, and twenty-six of more than 10 weeks. Of the four rats with hypertension of seven weeks or less duration three returned so nearly to their original blood pressure levels as to be indistinguishable from normal (viz., 1, 7, 13 mm. above normal) but probably this experiment must be regarded as one in which the number of nephrectomies during acute hypertension is insufficient for discussion in this connection.

It seems that the failure of these two groups to come to the same conclusion as we did with regard to the effect of nephrectomy early in hypertension, may in part depend upon our having used considerably

more rats, namely 21, in the duration group of from five to ten weeks.

The significance of the conclusion that this type of hypertension is initiated by the kidney but maintained by an extra-renal mechanism was not fully realized at the time we published these data. Later, Dr. Sapirstein pointed out to us that this was a key observation in the understanding of renal hypertension, but we were unaware that Pickering[23] had already arrived at the same conclusion on the basis of observations reported by Taggart and Drury[29] on the responses to renin of normal and hypertensive rabbits.

Pickering[23] discussed the evidence for the proposition that rabbits during the first week after their renal arteries are constricted have renin hypertension but that later a "non-renal factor" plays an important, and perhaps the chief, role in maintaining the raised pressure. He lists the following three points, and I quote his words:

"In the first week after constricting the renal artery, it has been here shown that the hypertension is usually abolished completely by removing the ischemic kidney. The time taken for the arterial pressure to fall to its normal level is of the same order when the ischemic kidney is removed as it is after stopping an intravenous infusion of renin lasting 4 hours. Finally the renin content of the ischemic kidney is at this stage increased. These facts indicate that the kidney is solely responsible for the hypertension and suggest that the release of renin from the ischemic kidney is the mechanism involved."

Removal of the hypertensive rabbit's kidney after seven weeks of clamping does not abolish the hypertension. He goes on to point out that this concept of a change from a renal to a non-renal mechanism is in harmony with the finding that the newly hypertensive rabbit has a raised renin content in its hypertensogenic kidney whereas the rabbit of longer hypertension does not.

Grollman[30] reported that in the rabbit (as in the rat) nephrectomy, after ten weeks of hypertension due to renal compression, does not abolish the hypertension.

In the dog there are no large series of experiments from which one can determine whether the duration of hypertension influences the remedial value of nephrectomy. In general, by selecting from the reported experiments those in which the duration of the hypertension is reported, one may conclude that removal of the kidney causing hypertension during the first month usually abolishes the hypertension.

If the kidney is removed at three months or later, the hypertension usually persists, but before accepting this evidence as a significant differentiation between acute and chronic hypertension in the dog one must consider carefully one well-documented experiment illustrated in Goldblatt's report.[40] This experiment shows that one dog with unilateral renal hypertension of nine months' duration was completely cured of its hypertension by ipsilateral nephrectomy. The evidence in this instance seems clear cut and the importance of this experiment is so great that it seems unfortunate that only one such animal was available and that so little time was allowed to guard against spontaneous reappearance of its hypertension.

Verney and Vogt[41] give data on two hypertensive dogs nephrectomized on the 15th and 20th days respectively which shows an immediate return of blood pressure to the original level. The authors speak of an additional 4 dogs which behaved similarly but do not give the data. There seems to be little doubt from these experiments and from those of several others (Blalock and Levy,[42] Rodbard and Katz,[43] and Goldblatt[40]) (Rodbard and Katz 6 animals from 5-28 days) that removal of the kidney from a unilaterally hypertensive dog abolishes the hypertension if the nephrectomy is done within the first few weeks.

These studies on nephrectomy in dogs, rats, and rabbits indicate clearly that renal hypertension is dependent upon the kidney only in its early stages but is later maintained by some other means.

The Role of the Nervous System: At Sapirstein's suggestion we (Reed, Sapirstein, Southard and Ogden[44] and Sapirstein and Reed[45]) gave rats during the early weeks of renal hypertension Nembutal, yohimbine or F883 with no effect upon their hypertension; but animals which had been hypertensive for a number of months usually showed a lowering of their blood pressure in response to these drugs. This suggests that the mechanism maintaining the later stages of hypertension involves the nervous system, particularly the sympathetic system, and that this nervous mechanism is not operating in early hypertension. The experiments of Dock and Dock and Rytand which demonstrate that pithing completely abolishes renal hypertension falls in with this suggestion.

Jacobs and Yonkman[46] gave yohimbine to four dogs hypertensive from silk perinephritis. The administration of the drug was started fifty to seventy days postoperatively about three to four weeks after

the hypertension was established. In one of these there was an unequivocal fall of blood pressure; in two more a fall was recorded but was so small as to leave its significance in doubt; in the fourth dog no change occurred. The dog whose blood pressure fell with certainty received its medication for twice as long as the dog which showed no change, and even so neither this nor any of the four showed blood pressure restored to the preoperative level. The method of administration of the drug, the species of animal and other differences, make these experiments difficult to compare with those of Reed, Sapirstein, Southard and Ogden, but Jacobs and Yonkman's results might be summarized thus:—administration of yohimbine to newly hypertensive dogs did not consistently lower the blood pressure and never abolished the hypertension. Stated in this form they confirm the observations of Reed, Sapirstein, Southard and Ogden on rats, but the distinction between "newly" and "chronically" hypertensive dogs cannot be made from Yonkman's data though it may be cautiously inferred from other observations and from the life-span of dogs. The concept that dogs of two months' hypertension are nearing or in the "chronic" category would fit in with the work of Friedman.[37]

The Effect of Sympathectomy on Experimental Hypertension: The experiments just described with Nembutal, yohimbine and F883 suggest that the extra-renal mechanism may be concerned with the sympathetic nervous system. If this is so sympathectomy performed early in the disease might be expected to be without effect whereas it might be expected to restore the blood pressure to its normal levels if performed after renin activity had ceased.

Sympathectomy on dogs with renal hypertension fails to restore the blood pressure to normal values. Generally, the duration of the hypertension before the performance of sympathectomy has not been stated, but where this information is available the sympathectomy has been performed early in the course of hypertension at a time when its failure to affect the disease is to be expected by comparison with the failure of sympatholytic drugs to cure experimental hypertension in rats. Heymans[11] removed the sympathetic nervous system with the exception of the innervation to the kidneys, raised the sympathetic tone by moderator nerve section and observed hypertension due to the vascular clamping of the renal arteries. In these dogs the kidneys were denervated six weeks later and the blood pressure was markedly lowered. This indi-

cates that at that time renin was solely responsible for the hypertension and that the nervous system was not participating except by producing renal vasoconstriction.

Verney and Vogt[41] by compression of the renal arteries in six dogs previously sympathectomized recorded moderate to great rises in blood pressure in every case. In one of their dogs the preoperative blood pressure was 116. It rose to a peak of 142 on the 48th day after renal artery compression and had declined to 122 by the 90th day. In another dog the pressures were preoperative 105, 46th day, 170. These dogs are the two dogs followed for the longest time according to their published data and therefore suggest that in the absence of the sympathetic nervous system hypertenson of renal artery origin does not persist beyond the third month.

A number of sympathectomies have been performed in hypertensive men but the data from these do not appear to be useful for this discussion for the following reasons:

First, there are many who doubt if hypertension of the Goldblatt type commonly occurs in man and none of the few human hypertensives whose disease seems clearly to be due to this mechanism have been subjected to sympathectomy.

Second, it is rarely possible to say with any certainty how long hypertension has existed in an individual coming up for sympathectomy; probably it is always more than the period corresponding to the few weeks we have been considering in animals.

Third, many of the series of sympathectomies in man are inadequately controlled both with respect to the careful evaluation of the effects of other therapeutic measures upon the blood pressure and with respect to adequate follow-up data.

For these reasons it seems best at the present time not to attempt to analyze and evaluate the various successful and unsuccessful sympathectomies reported with regard to their bearing on this hypothesis. Undoubtedly the recent practical interest in therapeutic sympathectomy will lead to the publication of series in which the data are adequate for use and in which follow-up information can later be effectively collected.

Similarly the facts that Gregory[47] lowered the blood pressure of hypertensive patients by means of spinal anesthesia and that he demonstrated the absence of effective renal pressor activity in his hypertensive

patients must be interpreted with caution until we can be sure whether these phenomena are true throughout the course of hypertension or if not at what stages they are present.

The effects of sympathectomy, the change in response to drugs, the change in the response to nephrectomy, the change in the sensitivity to injected renin, the change in the renin content in the kidney, and the change in the findings on testing for renin in circulatig blood are six basic evidences which may now be put together to offer a tentative story of the dual pathogenesis of experimental renal hypertension.

Dual Mechanism Stated: The application of the clamp, probably by diminishing the pulsatile expansion of the kidney causes the liberation of renin and elevates the blood pressure. This raised blood pressure brings into play an extra-renal (neurogenically operated and sympathetically mediated) mechanism which maintains the vasoconstriction. The liberation of renin ceases either because the general change in vascular dynamics starts the kidney moving again or else by some other adaptation or exhaustion of the renin mechanism. The cause for the cessation of renin liberation is unknown, but the important fact which must be realized is the existence of this very fundamental change in the mechanism of hypertension.

Many experiments would have yielded much more valuable information had this change of mechanism been taken into account in planning the experiments or had the exact intervals between the establishment of hypertension and subsequent experiments on the animal been routinely reported.

The present state of the hypothesis discussed is that no critical experimental data or series of clinical observations are incompatible with this theory as applied to hypertension of the Goldblatt type, but that the critical data to support it are insufficient and are based in part on rats, which are suspect. Many scattered observations point toward the correctness of this view but critical surgical and pharmacological experiments must be performed on dogs and other species before it can be regarded as established.

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TREATMENT OF ACUTE ANTERIOR POLIOMYELITIS WITH CURARE AND INTENSIVE PHYSICAL THERAPY*

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INTRODUCTION

A YEAR has passed since the first complete report was made at the Orthopedic Section of the New York State Medical Society[1] of a method of treating acute anterior poliomyelitis from the onset of the disease, with curare and intensive physical therapy. Therefore it is fitting that another report be made on the second year's experience with this method of treatment. This is the first complete year's experience with the method as an entity. The first report included much developmental work. In treating the patients seen in 1946, the regimen[2] which was described last year was followed meticulously. The results have been most gratifying.

STATISTICS

Fifty-two patients were treated from June 1, 1946 through April 1, 1947. There were three deaths. This is a mortality rate of 6 per cent. One of these was a fulminating Landry's type ascending paralysis. The other two died with what our pathologist described as acute pulmonary edema. This is probably of the neurogenic type as described by Henneman.[3] There are three fair results or 6 per cent. One is a patient who had a total quadriplegia with paralyzed abdominal muscles. There were numerous other factors in this patient, but her diagnosis finally was established as acute anterior poliomyelitis. She has recovered the use of her upper extremities and is beginning to show myographic evidence of power in her lower extremities. Her abdominal musculature is recovering nicely. The other two have major paralysis of muscles controlling one or more joints. It is to be noted here that the criterion

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on which results are based is the evaluation of muscle power, *NOT* functional ability. The vagaries of estimating muscle power are great enough. When function is used as the criterion, the margin of error is too great to allow dispassionate evaluation. One patient, or 2 per cent, is classified as good. She has no total paralysis and only moderate involvement of the anterior and middle deltoid. She had had a total paralysis of that extremity. There are four patients or 8 per cent classified as very good results. These have minor pareses that require a well trained observer to detect the weaknesses. Forty-three patients, or 83 per cent, have excellent results, which means that there are no residua of the disease. In re-evaluating the percentages on the basis of those who lived, 47 of the 51 patients show very good or excellent results. This is 92.1 per cent.

Physiology

The concepts on which this method of treatment is based are elementary physiologic and orthopedic principles. These are:

1) A muscle which has lost its normal length (a short muscle) is a weak muscle. Deformities result because of short muscles.

2) Long continued spasticity or spasm will cause a muscle to become shortened.

3) Pain may be an etiological factor in muscle spasm. It is not the principal causative agent of muscle spasm or spasticity.

4) Sherrington's law of reciprocal innervation must be in effect for normal muscle action.

5) Normal circulation is necessary for the physiological return of muscle power.

It is thought that these concepts are best maintained by the use of curare, muscle stretching, active exercises, and activity from the onset of the disease or from as soon after the onset of the disease as possible.

No attempt will be made at this time to discuss the electrophysiology or electropathology of muscles affected by acute anterior poliomyelitis. All of these patients have been followed carefully and thoroughly with electromyographic studies. It is felt that this subject has no place in this presentation. A separate report of these studies will be made at another time. From the experiences with myography, it is apparent that certain of the published concepts will have to be reviewed carefully and, in all probability, be revised.

Diagnosis and Dosages

When a patient is admitted to the Monmouth Memorial Hospital at Long Branch, New Jersey, for observation as a possible acute anterior poliomyelitis patient, the procedure is as follows: A complete history and physical examination are done. An effort is made to determine the presence of muscle paralysis or paresis. Each joint is put through its range of motion to determine whether or not there is any loss of muscle length. A spinal tap is performed and, while the spinal fluid is being examined in the laboratory, myographic studies are made. If the diagnosis of acute anterior poliomyelitis is confirmed, the patient is given the first injection of curare while attached to the myograph machine; 0.9 unit of curare (Intocostrin) per kilogram of body weight is administered intramuscularly every eight hours for the first twenty-four hours. After this period of time, the dosage is increased to 1.5 unit of Intocostrin per kilogram of body weight every eight hours until all evidence of muscle spasm or spasticity has disappeared.

It must be emphasized that curare is NOT a "one shot remedy." From the nature of the disease, it should not be expected that one dose of this drug could possibly alleviate or obliterate permanently the hyperexcitability of muscle which is present. Therefore, it is necessary to administer the drug repeatedly until the disease has burned itself out and normal, or relatively normal, muscle and nerve physiology reestablished.

Much has been said, written and implied as to the dangers of curare. At the Monmouth Memorial Hospital, approximately 10,000 intramuscular injections of curare have been given to patients with acute anterior poliomyelitis. Its pharmacologic antidote, physostigmine, has been administered twice. It is doubtful that it was necessary either time. There have been no fatalities nor ill effects attributable either directly or indirectly to the drug.

Physical Therapy

Physical therapy is instituted forty-five minutes after the first injection of curare. Each joint is put through a full range of motion or as nearly a full range of motion as possible at the time. This must be judicious stretching but pain is not the criterion of the amount of stretching necessary. The part is taken through its range of motion

until resistance is encountered. Then, with gradual pumping motions, that range of motion is increased as much as possible at that time. The amount of increase varies in each case, but it is a gradual increase, not an abrupt one which might result in torn muscle fibers, etc. *There never must be residual pain after stretching.* The stretching procedures are instituted three times after each daytime dosage of curare. The patients are taught to stretch themselves and one another. In addition to the stretching, active exercises are started immediately. Fundamental gymnastics of the Danish Group are utilized, stressing flexibility and coördination and getting strength coincidentally. It has been found that this approach to active exercises makes it more interesting for patients of all age groups. All activities are encouraged in order to keep the patients moving and busy. Occupational therapy, sports, tumbling, etc., form a part of the routine. Bed rest is discouraged except for febrile patients whose fevers are 102° or over. Mechanotherapy, hydrotherapy and electrotherapy are incorporated in the treatment.

Follow-up Care

The follow-up care of these patients after discharge from the hospital is of the utmost importance. Each patient is required to report back for physical therapy daily. These patients are examined by a member of the staff at least once a week. The number of treatments per week is decreased as rapidly as the patient's condition allows. Any time that the attending orthopedists find evidences of retrogression, i.e., return of spasm as evidenced by loss of muscle length or muscle strength, curare is administered again as an aid to the reëstablishment of normal muscle physiology. The physical therapy procedures, of course, have been continuous. All patients are examined at monthly follow-up clinics for at least the first year, when this is possible. It has not been possible to follow five of the patients. One, due to the fact that the family doctor frightened the mother about the after-effects of curare. One, because the grandparents insisted on prescribing the therapy. Two patients moved to Florida. The fifth patient was transferred to another institution. Her wedding announcement was received within eight months of this transfer.

Seventy-two patients, treated in 1945 and 1946. have been followed from the time of the onset of the disease to the present time with the following results:

1) No deformities have developed.

2) No braces have been used with the exception of a tongue spring for drop foot.

3) No crutches are being used.

4) Two patients use a cane occasionally.

Psychotherapy

Psychotherapy is a most important part of this program. Every effort is made to make the patients feel at home in the hospital. Their own clothes are brought to them as soon as the isolation period is over. Favorite playthings, hobbies, etc., are brought to them from their homes. Toys, games and books are supplied. There is a combination radio-victrola for their use. The hallway becomes a parking lot for tricycles. For the children, meals are served on a long picnic table with benches. Birthdays always are celebrated with parties. The atmosphere never is allowed to be that of a sick room. The confidence and coöperation of the patient must be obtained by the physical therapists, the occupational therapists, and the nurses. Certain standards of behavior in treating patients with acute anterior poliomyelitis have been established. These are, in brief, as follows:

1) Never lie to a patient. If the patient is going to be hurt, tell him so.

2) Never bribe a patient in an endeavor to get his coöperation.

3) Make it a practice to spend some time in recreation with the patients daily. This may consist of roughhouse, reading, or innumerable methods of having the patients do things under the guise of play. This is essential so that the nurses and therapists do not become ogres, but maintain normal personal relationships with the patients. This is just as important for the adults as for the children. It is a real program based on elementary psychological principles.

Conclusions

1) It is believed that the described method of treatment of patients with acute anterior poliomyelitis will accomplish the following: (a) Shorten the period of hospitalization. (b) Decrease the number of personnel necessary to care for the patients. (c) Give better functional results. (d) Prevent deformities.

2) No claim is made that this is a cure for acute anterior poliomyelitis, but it is asserted that it is a better method of treatment.

(*References on page 669*)

Nos.	Adm. #	Initial	Age	Pre-hosp.	Presenting Symptoms	Muscle Tightness	Muscles Involved	Type	Myo	Doses of Curare	Hosp. Days	Present Condition
1	80151	DA	16 mos.	5 days	Temp., restless unable to walk, labored breathing.	Neck.	Intercostals, gluteals, hamstrings, quadriceps, tibials and triceps, surae peroneals and abdominals.	B	V	311	112	VG
2	82695	CSA	11 yrs.	4 days	Temp., headache, pain in back and r. arm, vomiting.	Back, neck, quads, strings.	Right triceps.	S	V	142	54	E
3	81097	NB	2½ yrs.	7 days	Temp., stiff neck headache, nausea.	Opisthotonos, hamstrings, tibials.	limping l. leg. All leg muscles weak.	S	V	36	19	E
4	81677	RB	6 yrs.	15 days	Limping, seen in clinic.	Neck, back, hamstrings.	Neck flexors, l. deltoid, hamstrings and quads, r. leg.	S	V	67	39	E
5	82920	PB	18 mos.	4 days	Slight ataxia, r. leg limp.	Hamstrings, back.	R. tibialis, neck, flexors,	S	V	118	51	E
6	83735	RC	4 yrs.	2 days	L. leg limp (ref. from Fitkin).	Hamstrings, and back.	L. quads and hams, neck flexors.	S	V	216	85	E
7	83450	JC	7 yrs.	5 days & 5 on medical.	Unable to swallow.	R. shoulder, hamstrings, neck and back.	Tibialis, hamstrings, neck flexors. r. deltoid & triceps.	BS	V	111	58	E
8	80747	AC	9 yrs.	3 days	Cold, nausea, pain in neck & back, unable to stand or walk.	Hamstrings, back, neck.	Hamstrings, r. leg muscles, gluteals.	S	V	65	34	E
9	86169	DC	2 yrs.	3 days	Temp., sore throat, r. hip limp.	R. hamstrings & gastrocnemius.	Abdominals, neck flexors, r. hip flexors.	S	V	26	24	E
10	82474	HC	2 yrs.	2 days	Cold, unable to walk.	Hamstrings & back.	L. quads & hams.	S	V	57	27	E
11	80620	SD	3 yrs.	4 days	Chills, fever, unable to sit up unable to use r. arm.	Neck, hamstrings, back, r. arm.	Flail r. arm, abdominals.	S	V	500	193	G
12	80506	DF	6 yrs.	Ped 2 days	Cyanosis, twitching, temp. 103-105.	L. lower ext.	Intercostals, r. facial.	BE S	V	3	2	D
13	82241	CF	3 yrs.	3 wks.	Temp., nausea, unable to walk or stand.	Hamstrings, gastrocnemius, back.	Flail r. leg.	S	V	151	80	VG

Nos.	Adm. #	Initial	Age	Pre-hosp.	Presenting Symptoms	Muscle Tightness	Muscles Involved	Type	Myo	Doses of Curare	Hosp. Days	Present Condition
14	80918	KF	9 yrs.	10 days	Pain in thighs & limp, temperature.	Neck, left hamstrings, back.	Adductors l. and r., l. quadriceps and hamstrings.	S	V	90	37	E
15	81398	BG	6 yrs.	2 days	Headaches, temp., pain in neck, flail r. arm.	Hamstrings, back and neck flexors, r. arm.	R. arm—flail, r. tibial, gastroc., adbominals, quad.	S	V	225	109	VG
16	82319	JG	3 yrs.	3 days	Respiratory difficulty, unable to talk or swallow. Temp. 104.	Slight nuchal rigidity.	None.	B	No	2	1	D
17	83971	JG	2 yrs.	2 wks.	Cold, diarrhea, paresis l. leg, limps.	General.	L. hip & leg muscles all weak.	S	V	47	21	E
18	81220	LG	12 yrs.	5 days	Respiratory and swallowing difficulties, comatose.	General.	L. facial, intercostals and abdominals.	BE	V	106	41	E
19	82539	MG	18 yrs.	3 mos.	Fever, nausea, vomiting, paralysis l. arm.	General.	Deltoid (1) = 0.	S	V	84	36	E
20	81996	HH	16 yrs.	2 days	Paralysis lower exts.	General.	Complete paralysis.	AP	V	3	1	D
21	81304	EH	10 yrs.	8 days	Temp., swallowing difficulties, paralysis l. arm, stiff neck, nausea.	Hamstrings, neck and back, l. arm.	Flail l. arm, swallow muscles, abdominals.	BS	V	157	63	E
22	81305	HH	9 yrs.	4 days	Resp. difficulty swallowing difficulty.	Back, neck, hamstrings.	Abdominals, tibials, l. biceps & triceps.	BS	V	145	63	E
23	79490	LH	15 yrs.	16 days	Sore throat Weak arm (R)	Hamstrings Sacrospinalis.	R. upper ext. 3+	BS	V	50	33	E
24	84451	MJ	22 yrs.	3 days	Cold, sore throat, stiffness, unable to walk.	None.	None.	S	V	76	34	E
25	84726	FJ	3 yrs.	2 wks.	Ataxia, unable to stand or walk.	Hamstrings, back and neck, heel cords.	R. leg muscles.	S	V	107	37	E
26	81291	AK	10 yrs.	6 days	Stiff neck, nausea, pain in legs. Unable to stand or sit. Headache, diarrhea, cramps.	General.	Flail r. arm and leg. Weak l. arm & leg. Abdominal leg flexors.	S	V	410	144	VG

Nos.	Adm. #	Initial	Age	Pre-hosp.	Presenting Symptoms	Muscle Tightness	Muscles Involved	Type	Myo	Doses of Curare	Hosp. Days	Present Condition
27	81139	GL	5 yrs.	2 days	Pain in l. knee and thigh.	Neck & back quadriceps. L. hamstrings.	Abdominals. L. iliopsoas. L. quadriceps.	S	V	50	19	E
28	83893	RL	7 yrs.	4 days	Pain in left ear. Cold, unable to walk on l. leg.	Back and hamstrings.	Abdominals and quads.	S	V	47	32	E
29	82616	WL	10 yrs.	2 days	Nausea, pain in neck, back and legs.	Hamstrings, sacrospinalis.	Abdominals.	S	V	65	27	E
30		HM	7 yrs.	1 day	Pain in abd. about unbilicus.	Opisthotonos.	Neck flexors. Abdominals.		V			
31	81761	JM	8 yrs.	11 days	Sore throat, stiff neck flail R. arm.	Back, hamstrings, wrist & finger flexors.	Flail arm (R).	S	V	189	68	G
32	81173	RMcG	13 yrs.	3 days	Nausea, sore throat, headache, stiff neck and back.	Opisthotonos in general.	L. iliopsoas and hamstrings, triceps.	S	V	140	51	E
33	81377	AM	35 yrs.	3 days	Vomiting, cold, headache, pain in knee, paralysis l. arm & leg, weakness r. arm & leg.	General.	L. arm and leg, abdominal, weakness r. arm and leg.	S	V	135	50	E
34	83460	JM	6 yrs.	2 days	Pain in legs, limps on r.	Hamstrings, back & back.	R. quads & hip abductors.	S	V	54	34	E
35	83730	EM	3 yrs.	3 days	Fever, sore throat, swallowing difficulty (convulsions for years).	Hamstrings, neck and back.	None.	BE	V	105	193	E
36		JN	5 yrs.	?	Pain in neck, fever.	Nuchal rigidity. paraspinal.	None.	S	No	None.	3	E
37	86706	JP										
38	82318	PP	3½ yrs.	2 days	Pain in neck and back.	Pectorals, hamstrings, back and neck.	Abdominals and neck flexors.	S	V	39	20	E
39	83231	SP	10 mos.	4 days	Sore throat & stiff neck.	Hamstrings, neck, back, tibials.	Flail r. leg.	S	V	221	76	VG
40	81257	DP	27 yrs.	3 wks. other hosp.	Unable to move. Breathing and swallowing difficulty.	General.	Quadriplegia, abdominals.	BS	V	517	249	F
41	82512	WR	5 yrs.	3 days	Abdominal cramps, Temp., pain r. leg. Unable to walk.	Neck, back, hamstrings, quadriceps.	Abdominals, neck flexors, muscles of r. leg.	S	V	162	79	VG

Nos.	*Adm. #*	*Initial*	*Age*	*Pre-hosp.*	*Presenting Symptoms*	*Muscle Tightness*	*Muscles Involved*	*Type*	*Myo*	*Doses of Curare*	*Hosp. Days*	*Present Condition*
42	80183	RR	12 yrs.	4 days	Fever, malaise, vomiting, pain in back and both legs.	Neck and back, hamstrings, quadriceps.	Peroneals and toe flexors.	S	V	138	50	E
43	83331	WT	26 yrs.	3 wks.	Headache, fever, unable to move, painful eyes.	Gastrocs, hamstrings, back and quadriceps.	Quads and tibials.	S	V	70	35	E
44	86240	WV	2 yrs.	2 days	Fever, pain in legs & arms, r. hip limp.	None (?)	Abdominals, neck flexors, hip flexors.	S	V	33	26	E
45	81015	JV	5½ yrs.	4 days	Fever, sore throat, pain in neck, stiff neck and back.	Neck, back, hamstrings.	Neck flexors, abdominals & serratus.	S	V	48	23	E
46	81252	CW	5½ yrs.	8 days	Fever, sore throat, pain in neck, weakness.	Neck, back, gastrocs & hamstrings.	Neck flexors, abdominals.	S	V	67	27	E
47	80607	WW	5 yrs.	4 days	Pain in legs, stiff neck, fever.	Neck and back, hamstrings & gastrocs.	Abdominals, neck flexors & tibials.	S	V	138	54	E
48	*	SMcN	11 yrs.		Pain in back and leg. Walking with a limp.	Neck and back, hamstrings & quadriceps, gastroc. & tibial.	Back and right leg.	S	No	78	81	E
49	*	CMcN	13 yrs.		Pain in back.		Back muscles.	S	No			E
50	*	H	14 yrs.		Pain in leg, unable to walk.		Right leg.	S	No	42	18	E
51	*	IH	22 yrs.		Opisthotonos, pain in back, headache, pain in chest.	Neck & back, hamstrings, & pectorals.	Neck flexors and back muscles.	S	No	57	61	E
52	*	RS	4 yrs.		Pain in l. arm and unable to use it.	General.	Biceps, triceps & deltoid.	S	No	45	20	E

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